CLINICAL PROFILE OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION

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Dedicated

to

my Family

&

Teachers

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ABBREVIATIONS

ADA	Adenosine deaminase
AFB	Acid fast bacilli
ATT	Antitubercular therapy
BMI	Body mass index
CA	Carcinoma
CBC	Complete blood count
CBNAAT	Cartridge based nucleic acid amplification test
CDC	Centers for disease control and prevention
CECT	Contrast enhanced computed tomography
CKD	Chronic kidney disease
CLD	Chronic liver disease
CT	Computed tomography
CXR	Chest x ray
DAMA	Discharge against medical advice
DILI	Drug induced liver injury
DM	Diabetes mellitus
ESR	Erythrocyte sedimentation rate
E.COLI	Eschrechia coli
HBV	Hepatitis b virus
HCV	Hepatitis c virus
HIV	Human immunodeficiency virus
HRCT	High resolution computed tomography
ICD	Intercostal drainage
i.e	That is

K.pneumoniae	Klebsiella pneumoniae
LDH	Lactate dehydrogenase
LFT	Liver function test
LMWH	Low molecular weight heparin
MRSA	Methicillin resistant staph aureus
NPV	Negative predictive value
PCD	Percutaneous drainage
PCR	Polymerase chain reaction
PPV	Positive predictive value
P.aeruginosa	Pseudomonas aeruginosa
RA factor	Rheumatoid arthritis factor
RFT	Renal function test
RTPCR	Reverse transcriptase polymerase chain reaction
SGOT	Serum glutamic oxaloacetic transaminase
SGPT	Serum glutamic pyruvic transaminase
TB	Tuberculosis
TLC	Total leukocyte count
TSH	Thyroid stimulating hormone
USG	Ultrasonography
VATS	Video assisted thoracoscopic surgery

Abstract

INTRODUCTION: The pathological accumulation of pleural fluid is very common. Based on light's criteria pleural effusion is classified as exudative or transudative. In this study we will focus on the clinical profile of exudative pleural effusion and know the outcome wherever possible.

AIM OF STUDY: To study the clinical profile of exudative pleural effusion and know the outcome.

MATERIALS AND METHODS: The study was carried out on 100 in-hospital patients aged > 18 years diagnosed with exudative pleural effusion as per the Light's criteria in Dayanand medical college and hospital. Diagnostic/therapeutic guided thoracentesis was done.They were followed at 1 month and 3 month of discharge.They were classified into various etiologies as per the test criteria/methodology. Their demographic, etiological, clinical, radiological profile and outcome data were recorded and analyzed.

RESULTS:100 patients with exudative pleural effusion were studied. 73% were males and 27% were females. The most common age group was 40-60 years. The most common presenting complaints were shortness of breath, fever, cough, chest pain, expectoration, chest trauma and hemoptysis.Tuberculosis was the leading cause of exudative effusion (42%) followed by parapneumonic (16%), empyema (15%), malignant (9%), other causes (11%) and unknown 7%. Lymphocyte to neutrophil ratio and ADA combined has sensitivity of 74.51% and specificity of 91.84% in diagnosing tuberculous effusion. MRSA, *E.coli*, *Klebsiella pneumoniae,* (*Pseudomonas aeruginosa), Acinetobacter*, *Burkholderia* were the common organisms isolated in empyema and parapneumonic groups. Adenocarcinoma lung was the most common etiology in malignant effusions.Other causes include pulmonary embolism, hemothorax, parasitic pleural effusions, viral and chylothorax. Chest tube insertion and/or fibrinolytic therapy were required for better drainage as per the indication in 50% of the cases. Overall mortality was 13%, empyema (4%) being the most common cause followed by malignant (3%) and parapneumonic effusions (3%), 'other' causes (2%) and (1%) tubercular group.

CONCLUSION: Determining the aetiological and clinical pattern of pleural effusion helps in optimizing diagnostic and therapeutic approach. Most of the exudative effusions resolve with treatment. High mortality is seen in empyema and malignant pleural effusion groups.

Keywords: exudative effusion, light's criteria, clinical profile, etiology, outcome.

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INTRODUCTION

A pleural effusion is collection of fluid abnormally present in the pleural space, usually resulting from excess fluid production and/or decreased lymphatic absorption.[1] Although a number of chemical tests have been proposed to differentiate pleural fluid transudates from exudates, the test first proposed by Light et al have become the criterion standards.[2] Exudative pleural effusions meet at least one of the following criteria, whereas transudative pleural effusions meet none (Light's criteria)[3] Pleural fluid protein divided by serum protein greater than 0.5. Pleural fluid LDH divided by serum LDH greater than 0.6. Pleural fluid LDH greater than two thirds of the upper limit of normal serum LDH.[3] Transudates are caused by a small, defined group of etiologies[3], diagnosis of which is fairly easy as the underlying cause is clearly evident through history, clinical examination, and few common laboratory investigations.[4] However the same is not true in case of exudative pleural effusion. It has a wide variety of etiologies like infective (bacterial, tubercular, viral, fungal, miscellaneous), malignant, autoimmune, gastrointestinal, renal, pulmonary embolism, cardiac, traumatic and miscellaneous causes. Hence determination of exact etiology can be quite challenging for a physician. Despite this, a considerable percentage of pleural effusions go undiagnosed. The etiological distribution of pleural effusions depends on the geographical area, patient's age, and advances in the diagnostic methods and treatment of the underlying causes. Patients can present with complaints of pleuritic chest pain, shortness of breath and cough which is mostly non productive. Other symptoms include fever, hemoptysis, malaise, weight loss, generalized weakness and symptoms specific to etiology of the pleural effusion. Physical examination aids in clinical diagnosis of pleural effusion which includes general physical examination and systemic examination. Dullness to percussion, decreased tactile fremitus, and asymmetrical chest expansion, with

diminished or delayed expansion on the side of the effusion. These are the most reliable physical findings of pleural effusion.[5,6]

Diagnosing a pleural effusion includes radiologic tests like chest x ray, ultrasound chest and CECT chest may be needed sometimes. Chest X ray is the first investigation to be ordered which can accurately tell the presence of pleural effusion. At least 175 ml of pleural fluid should be present for costophrenic angle blunting. However 75 ml is sufficient to detect costophrenic angle blunting on lateral films. Larger pleural effusion can cause mediastinal shifting to the contralateral side. If mediastinal shifting is towards the same side as pleural effusion, bronchoscopy is done to look for endobronchial neoplasm and assess the patency of the bronchial tree. When the diagnosis of pleural effusion is not clear on chest radiography, ultrasound chest is done. It can detect the presence of pleural fluid as small as 3-5 ml. One can describe presence of septations and loculation, differentiate between pleural fluid and pleural thickening. Also ultrasound guided diagnostic thoracentesis can be done. For pleural fluid drainage, ultrasound guided pigtail can be inserted. CT chest is excellent at detecting small amounts of pleural fluid and is also often able to identify the underlying intrathoracic causes (pleural deposits or primary lung neoplasms) as well as subdiaphragmatic diseases (subdiaphragmatic abscess). CT scan is the modality of choice for further assessment of pleural effusios and demonstration of homogeneity of pleural masses and areas of fatty attenuation or calcification.[7] After informed consent pleural fluid diagnostic aspiration is done either directly or under ultrasound guidance and a needle is inserted into pleural space to aspirate fluid from around the lung. It can lead to some major complications like pneumothorax, bleeding, re-expansion pulmonary edema, hypotension, hemothorax, puncture of the spleen or liver and vasovagal syncope. It is a routine practice to obtain chest x-ray post procedure. Post procedure chest x ray should be done if the patient is ventilated, air is aspirated,

needle inserted more than once or symptoms and signs of pneumothorax develop. These complications can be fatal if not adequately managed.

Once the pleural fluid is aspirated, it is first visually inspected for its color, presence of turbidity, centrifugation is done for differentiating into chylothorax and infective etiology. It is sent for laboratory evaluation which include biochemical, pathological, cytological and microbiological analysis. Some special tests like immunological studies, triglyceride levels, rheumatoid factor, antinuclear antibody and amylase levels can be performed if indicated clinically. Pleural fluid is first classified into transudative and exudative using Light's criteria as already discussed above. Clinical judgment is required when pleural fluid test results are near the cutoff values as Light's criteria misidentify approximately 20-25% of transudates as exudates. Serum minus pleural protein concentration level of less than 3.1 g/dL, instead of a serum/pleural fluid ratio of greater than 0.5, more correctly identifies exudates in these patients.[8] Pleural effusion can remain undiagnosed after initial workup. In these cases, more invasive procedures are performed like bronchoscopy (when patient has parenchymal abnormalities or hemoptysis) surgical procedures like video-assisted thoracoscopy (pleuroscopy) and open thoracotomy (reveals an etiology in 92% of effusions with an operative mortality of less than 0.5%), medical thoracoscopy (may be diagnostic and therapeutic; complete drainage of the effusion and talc sclerosis can be performed at the time of the procedure). Pleural biopsy is considered when tubercular or malignant etiology is suspected. The yield is increased by taking multiple samples using CT-guided cutting-needle biopsy to detect specific abnormal areas, particularly for malignancy. Closed blind pleural biopsy is not recommended now as the yield is better with image-guided pleural biopsy or thoracoscopy. Single sample is usually sufficient to diagnose tuberculosis.

Treatment and management depends on the underlying etiology of pleural effusion. Pneumonia, malignancy, and TB cause most of the exudative

pleural effusions. Complicated parapneumonic effusions and empyemas should be drained. Malignant effusions are drained to palliate symptoms and pleurodesis may be needed to prevent recurrence. Of the common causes for exudative pleural effusions, parapneumonic effusions have the highest diagnostic priority. The duration of antibiotic therapy depends on many factors, for example, the sensitivity of the organism, extent of pulmonary parenchymal and pleural disease, response to initial therapy and adequacy of drainage.[9] Malignant effusions signify incurable disease with high morbidity and dismal mean survival of less than one year. Repeated thoracentesis, pleurodesis, or placement of indwelling tunneled catheters may be required for symptom palliation. Pleural effusions related to connective tissue disorders like rheumatoid arthritis and systemic lupus erythematosus are treated with steroids and resolution occurs approximately within two weeks of initiation of treatment.[10] Tubercular pleural effusions are treated with antitubercular therapy and steroids are also given in some patients. Antibiotics are given as per the underlying infective etiology. Patients with chylothorax require nutritional support with a diet rich in low-fat, medium-chain triglycerides which are absorbed directly into the portal circulation to reverse hypovolemia, immunosuppression, and protein and electrolyte deficiencies.[10] Therapeutic thoracentesis is indicated whenever a patient has developed respiratory distress. Pleurodesis is indicated when there is recurrent pleural effusion. Either intercostal drainage or pigtail catheter insertion can be done. Decortication, pleurectomy, pleuropneumonectomy, closure of bronchopleural fistula with or without grafting, window operation, fenestration surgery, thoracostomy, and thoracoplasty are the various surgical modalities available.[10]

AIMS AND OBJECTIVES

- To study the clinical profile of patients with exudative pleural effusion.

- To study the outcome of patients with exudative pleural effusion

REVIEW OF LITERATURE

A pleural effusion is collection of fluid abnormally present in the pleural space usually resulting from excess fluid production and/or decreased lymphatic absorption.[1]

ANATOMY AND PHYSIOLOGY

Pleural space is potential space between visceral and parietal pleura. The mean rate of both the production and the absorption of pleural fluid is normally 0.2 mL/kg/hr, which implies that the entire volume of the pleural fluid normally turns over within one hour.[11] There is equilibrium between production and resorption of pleural fluid in healthy individuals.

EPIDEMIOLOGY AND DEMOGRAPHY

The estimated prevalence of pleural effusion is 320 cases per 100,000 people in industrialized countries, with a distribution of etiologies related to the prevalence of underlying diseases.[12] A Study conducted by Saha K and et al in 2017 in West Bengal on demographic , clinical , biochemical and etiological characteristics of malignant pleural effusions from Eastern India identified that out of 166 patients, 72.89% were males and 7.11% were females. Mean age of presentation among males was 64.3 +/-12.7 and among females was 52.5 +/- 14.8. Most common presenting symptom was dry cough (87.9%) and the most common presenting sign was clubbing (54.5%). Massive effusion was found in 45.78% of cases.[13]

ETIOLOGY

There are many causes for exudative pleural effusion and it needs immense workup to know the underlying cause. Despite this, many exudative pleural effusions remain undiagnosed which is challenging as this directly affects the treatment. There are a number of mechanisms in the genesis of pleural effusion. Mechanisms of exudate formation include pleural or parenchymal inflammation, impaired lymphatic drainage, transdiaphragmatic movement of fluid from the peritoneal space, altered permeability of pleural membranes, and/or increased capillary wall permeability or vascular disruption. Pleural membranes are involved in the

pathogenesis of the fluid formation. Of note, the permeability of pleural capillaries to proteins is increased in disease states with elevated protein content. Various causes of exudative pleural effusion includes: neoplastic, infectious (tubercular, bacterial, viral, parasitic, fungal), pulmonary embolisation, gastrointestinal causes (pancreatitis, subphrenic abscess, intrahepatic or intrasplenic abscess, esophageal perforation, diaphragmatic hernia), heart diseases (post coronary artery bypass graft surgery, Dressler's syndrome, pericardial disease, pulmonary vein stenosis,post catheter ablation of atrial fibrillation), obstetric and gynecologic diseases (Ovarian hyperstimulation syndrome, fetal pleural effusion, postpartum pleural effusion, meigs' syndrome, endometriosis), connective tissue disorders (rheumatoid pleuritis , systemic lupus erythematosus, sjögren's syndrome, familial mediterranean fever, churg-Strauss syndrome, wegener's granulomatosis), drug induced pleural diseases (nitrofurantoin, ergot drugs, amiodarone, procarbazine, methotrexate, clozapine) and miscellaneous causes (asbestos exposure, post lung transplant, post bone marrow transplant, yellow nail syndrome, sarcoidosis, uremia, trapped lung, therapeutic radiation exposure, drowning, amyloidosis, milk of calcium pleural effusion, electrical burns, iatrogenic pleural effusion). A prospective study conducted by PP Prabhudesai and et al in Maharashtra (1993) on exudative pleural effusions in patients over forty years of age –analysis of seventy six patients indicated that malignant pleural effusions were the most common in this series, found in 49 patients (64.47%), all but one being metastatic from elsewhere. Forty were secondary to a carcinoma of a bronchus, 3 from carcinoma of breast, 1 each from the carcinoma of ovary, esophagus, and larynx; lymphoma accounted for the remaining 2. Infective causes accounted for 24 of the effusions (31.57%). Of the infections, tuberculosis was the most common, accounting for 17 of the 24. Other infective causes included bacterial empyema in four, ruptured amoebic liver abscess in two, and actinomycosis in one. Pancreatitis,

pulmonary thromboembolism, and a post-craniotomy syndrome were diagnosed in one patient each, while the diagnosis remained unknown in the remaining five patients. In two patients the diagnosis was made on autopsy.[14]

CLINICAL EVALUATION

Detailed history, physical examination, lab investigations and imaging is required to reach the final diagnosis.

HISTORY

Clinical symptoms are mainly dependent on the underlying mechanism of pleural effusion. Patients can even be asymptomatic at presentation. Dyspnea is a major, although nonspecific, respiratory symptom which accompanies pleural effusion, commonly with progressive worsening.[15.16] This may be relieved with therapeutic thoracentesis. However, shortness of breath may occur due to underlying lung parenchymal disease rather than pleural effusion. Pleuritic chest pain can occur and signify parietal pleural involvement and underlying exudative nature of pleural effusion. Dry cough is the third symptom. Extrapulmonary symptoms like weight loss, night sweats, fever, hemoptysis etc may be present. S Laxma Reddy and et al conducted a study published in 2019 named Clinical and etiological profile of an exudative pleural effusion in a tertiary care center at Hyderabad, Telangana. It was found that the most common presenting symptom was dyspnea (84%), followed by cough (80%), fever (65%), and chest pain (43%). The most frequent cause of pleural effusion was tuberculosis in 38% of patients, followed by parapneumonic effusion (28.5%) and malignant pleural effusion (22.2%).[17]

PHYSICAL EXAMINATION

Dullness to percussion, decreased tactile fremitus, and asymmetrical chest expansion, with diminished or delayed expansion on the side of the effusion are the most reliable physical findings of pleural effusion.[5,6] Mediastinal shift when pleural effusion is more than 1000 ml, inaudible breath sounds on auscultation, egophony at superior most part of effusion,

pleural friction rub are other physical findings on examination. Extrapulmonary manifestations aid in diagnosis of pleural effusion.

RADIOLOGICAL INVESTIGATIONS

After history taking and physical examination, it is important to confirm and document the pleural effusion radiologically. Collins et al.[18] injected fluid into the pleural spaces of upright cadavers. They demonstrated that at least 175 mL pleural fluid had to be injected before the lateral costophrenic angle was blunted, and in some cases, more than 500 mL pleural fluid could be present without blunting the lateral costophrenic angle. At times, for unknown reasons, substantial amounts of pleural fluid (more than 1,000mL) can be present and may remain in an infrapulmonary location without spilling into the costophrenic sulci or extending up the chest wall. Such pleural fluid accumulations are called *subpulmonic* or *infrapulmonary pleural effusions*. Although the posterior costophrenic angle is usually blunted, at times it is perfectly clear.[19] Lateral decubitus films more reliably identify smaller pleural effusions. It is now less carried out when bedside ultrasonography is present. With the help of ultrasound one can locate, quantify and distinguish between pleural fluid and pleural thickening. Note of septations or loculation can be made. Ultrasound is used to identify the site and ascertain the depth of pleural effusion, therefore increasing the safety of diagnostic and therapeutic thoracentesis. Ultrasound can also be used for inserting percutaneous catheters and performing pleural biopsy. Ultrasound is being used frequently to assess trauma patients for the presence of a hemo-thorax and/or a pneumothorax. Pleural tap can be done either based on the chest radiograph and located 1-2 rib interspaces below the level of dullness to percussion on physical examination or using ultrasound, a pleural tap site can usually be quickly identified and marked. The post procedure chest x-ray should be obtained. Complications related to the procedure include: Pain, pneumothorax, seeding of malignant tissue, bleeding, re-expansion pulmonary edema and adverse reaction to

anesthetic agent. The frequency of complications from thoracentesis may be lower when a more experienced clinician performs the procedure plus ultrasound is used for the procedure.

COMPUTED TOMOGRAPHY

CT chest is effective in detecting abnormalities in the lung parenchyma that can be missed on chest x ray. CT chest differentiates empyema with air-fluid level from lung abscess, identifies benign and malignant effusion.

PLEURAL FLUID LABORATORY ANALYSIS

Normal pleural fluid is a clear ultrafiltrate of plasma that originates from the parietal pleura, with a pH of 7.60-7.64, protein content of less than 2% (1-2 g/dL), less than 1000 white blood cells (WBCs) per cubic millimeter, Glucose levels similar to that of the plasma and Lactate dehydrogenase (LDH) less than 50% of plasma.

APPEARANCE

If pleural fluid appears bloody, one should get pleural fluid hematocrit . The blood in the pleural fluid is not significant if the pleural fluid hematocrit is less than 1%.[20] If the pleural fluid hematocrit is greater than 1%, the patient most likely has malignant pleural disease, a pulmonary embolus, or a traumatically induced pleural effusion.[20] If hematocrit is more than 50 % of peripheral blood hematocrit, diagnosis of hemothorax is confirmed and tube thoracostomy should be performed. When fluid is turbid or milky, centrifugation is performed and the supernatant of pleural fluid is examined. If the turbidity clears, pleural fluid is likely infectious, if it persists, differentials of chylothorax and pseudochylothorax should be on the list.

BIOCHEMICAL ANALYSIS

Biochemical analysis of pleural fluid is done to classify it into either exudative or transudative. Light et al. subsequently demonstrated that with the use of simultaneously obtained serum and pleural fluid protein and lactic acid dehydrogenase (LDH) values, 99% of pleural effusions could be correctly classified as either transudates or exudates.[2] Exudative pleural

effusions meet at least one of the following criteria, whereas transudative pleural effusions meet none (Light's criteria):[11]

1. Pleural fluid protein divided by serum protein greater than 0.5
2. Pleural fluid LDH divided by serum LDH greater than 0.6
3. Pleural fluid LDH greater than two thirds of the upper limit of normal serum LDH

Light's criteria identify nearly all exudates correctly, but misclassify approximately 20-25% of transudative effusions into exudative effusions, usually in patients on long-term diuretic therapy.[21] Using the criterion of serum minus pleural protein concentration level of less than 3.1 g/dL, rather than a serum/pleural fluid ratio of greater than 0.5, more correctly identifies exudates in these patients.[8] A study conducted by Rohit Rungta and et al in 2013 on Comparative analysis of pleural fluid biochemical parameters with cholesterol to differentiate transudates from exudates suggested that, according to their etiology, 7 of the effusions were transudates and 49 were exudates. Using a cutoff point of 45 mg for pleural cholesterol and values for protein and lactate dehydrogenase (LDH) of Light et al., the best diagnostic power corresponded to the combination of pleural cholesterol and LDH; cholesterol level over 45 mg/dL and/or LDH over 200 U/L differentiate exudates from transudates with a sensitivity of 99% and a specificity of 98%.[22]

PROTEIN

Pleural fluid protein level does not help in differentiating between various etiologies of exudative pleural effusion. At times, a pleural fluid meets the exudative criteria with its LDH, but not with its protein level. Such exudative pleural effusions are almost always parapneumonic effusions or are secondary to malignant pleural disease.[2]

LACTIC ACID DEHYDROGENASE (LDH)

As discussed earlier LDH is used to differentiate between exudative and transudative pleural effusion. LDH is a reliable indicator of the degree of pleural inflammation. Increasing levels of LDH in pleural fluid on repeated thoracentesis indicates serious inflammation and one should aggressively seek diagnosis. The presence of blood in pleural fluid does not significantly affect pleural fluid LDH measurement.

GLUCOSE AND Ph

Glucose measurement helps in making differential diagnosis of pleural fluid as low glucose (less than 60 mg/dl) is present in four main conditions namely parapneumonic effusion, malignant disease, rheumatoid disease, or tuberculous pleuritis. When the pleural fluid pH is used as a diagnostic test, it must be measured with the same care as arterial pH. In general, the pleural fluid with low glucose (<60 mg/dL) also have a low pH (<7.20) and high LDH levels[23]. If the pleural fluid pH is below 7.0, the patient invariably has a complicated parapneumonic effusion, and attempts should be made to remove all the pleural fluid with therapeutic thoracentesis or tube thoracostomy.[24]

AMYLASE

Pleural fluid levels above the normal serum amylase levels indicate a patient may have one of these: pancreatic disease, malignant tumor, or esophageal rupture.[25] However, it is not cost effective to obtain an amylase measurement on every undiagnosed pleural fluid; rather, pleural fluid amylase levels should be determined only when esophageal rupture or pancreatic disease is suspected.[26]

ADENOSINE DEAMINASE (ADA)

ADA levels tend to be higher in tuberculous pleural effusions than in other exudates. The two main diseases that cause an elevated ADA in addition to tuberculosis are rheumatoid pleuritis and empyema.[27,28] If the diagnostic criteria for tuberculous pleuritis also include a pleural fluid lymphocyte-to-neutrophil ratio greater than 0.75, the specificity of the test

is increased.[29] High pleural fluid ADA levels have also been reported with a very small percent of other neoplasms,[30] with Q fever[31] and with brucellosis.[32] ADA value above 40u/l is suggestive of pleural tuberculosis. If it is more than 70u/l and the patient does not have empyema and rheumatoid arthritis then it is diagnostic of tuberculous pleuritis. A study conducted in West Bengal (2018) by Maikap MK, et al., on Etiology and clinical profile of pleural effusion identified that the most common cause of pleural effusion in this study was tuberculosis (68.8%), and followed by malignancy (14%), empyema (6%), and transudative effusion (2.8%). Pleural effusion was commonly seen in males (66%).the occurrence of pleural effusion was maximum in the age group 21-30 years, but malignant pleural effusion was more common above 60 years of age. Right sided effusions were more common. Estimation of pleural fluid deaminase plays a significant role in the diagnosis of tubercular pleural effusion. Pleural fluid cytology and closed pleural biopsy can diagnose most of the cases of pleural effusion due to malignancy.[4]

LIPID STUDIES

When pleural fluid appears milky or turbid, one must centrifuge it. After centrifugation, if the supernatant is clear, it is empyema. If milky or cloudy then it is chylous or chyliform effusion. If the pleural fluid triglyceride level exceeds 110 mg/dL, the patient probably has a chylothorax; if the triglyceride level is below 50 mg/dL, the patient does not have a chylothorax. If the triglyceride level is between 50 and 110 mg/dL, the patient may or may not have a chylothorax.[33] When the diagnosis is uncertain, lipoprotein analysis of the fluid should be ordered. Malignancy is the most common cause of chylothorax particularly lymphoma (non hodgkins more than hodgkins). Iatrogenic injury to the thoracic duct has been reported with thoracic procedures. In particular, cardiothoracic surgery has been associated with 69-85% of cases of chylothorax in children. Demonstration of chylomicrons in the pleural fluid with lipoprotein analysis is diagnostic of chylothorax. A pseudochylothorax occurs due to

the accumulation of a cholesterol crystal-rich fluid within the pleural space in chronic existing effusion. Common causes of pseudochylothorax are chronic rheumatoid pleurisy, tuberculosis and poorly treated empyema.

PATHOLOGICAL ANALYSIS

RED CELL COUNT

Leakage of only 1 ml blood will impart red color to pleural fluid. One should obtain pleural fluid hematocrit and compare it with peripheral blood hematocrit; if pleural fluid hematocrit is more than 50% of peripheral blood hematocrit, then it is a hemothorax and chest tube should be inserted. If less than 50%, possible diagnosis include one of the following- malignancy, traumatic tap or pulmonary embolism.

WHITE BLOOD CELLS AND DIFFERENTIAL WHITE BLOOD CELL COUNT

WBC count alone is not of much significance unless differential wbc count is done. NEUTROPHIL - Predominance of neutrophils in pleural fluid indicates acute inflammation of pleura which can be seen in pneumonia, pancreatitis, pulmonary embolization, subphrenic abscess, and early tuberculosis. LYMPHOCYTES- Presence of more than 50% lymphocytes in pleural fluid indicates that a patient might have one of the following- malignancy, tuberculosis or a pleural effusion developing post coronary artery bypass graft surgery. Other differentials include lymphoma, sarcoidosis, chronic rheumatoid pleurisy, yellow nail syndrome, and chylothorax. EOSINOPHIL - Presence of either blood or air can cause pleural fluid eosinophilia. If neither is present, several unusual diagnoses should be looked for like asbestos-related pleural effusions, eosinophilic pneumonia, drugs like dantrolene, bromocriptine, and nitrofurantoin, parasitic diseases, Churg-Strauss syndrome. The origin of approximately 40% of eosinophilic effusions is not established, and these effusions may resolve spontaneously. Basophils and macrophages are not of much clinical significance.

CYTOLOGICAL EXAMINATION FOR MALIGNANCY

Pleural fluid should be tested for presence of any malignant cells especially when clinically suspected, exudative lymphocytic effusion and bloody effusion. The accuracy of the cytologic diagnosis of malignant pleural effusions has been reported to be anywhere between 40% and 87%. Heparinized samples (1 mL of 1:1000 heparin per 50 mL of pleural fluid) should be submitted for analysis if the pleural fluid is bloody and they should be refrigerated if samples are not to be processed within one hour. Diagnostic yield is increased when at least three different samples are sent to an experienced cytologist. A prospective study conducted by PP Prabhudesai and et al in Maharashtra (1993) on exudative pleural effusions in patients over forty years of age –analysis of seventy six patients indicated that malignant pleural effusions were the most common in this series, found in 49 patients (64.47%), all but one being metastatic from elsewhere. Forty were secondary to a carcinoma of a bronchus, 3 from carcinoma of breast, 1 each from the carcinoma of ovary, esophagus, and larynx; lymphoma accounted for the remaining 2. Infective causes accounted for 24 of the effusions (31.57%). Of the infections, tuberculosis was the most common, accounting for 17 of the 24. Other infective causes included bacterial empyema in four, ruptured amoebic liver abscess in two, and actinomycosis in one. Pancreatitis, pulmonary thromboembolism, and a post- craniotomy syndrome were diagnosed in one patient each, while the diagnosis remained unknown in the remaining five patients. In two patients the diagnosis was made on autopsy.[14]

MICROBIOLOGICAL ANALYSIS

GRAM'S STAIN AND AFB SMEAR

Gram's stain of exudative pleural effusion is performed. Routine smears for mycobacteria are not indicated because they are almost always

negative, unless the patient has a tuberculous empyema or unless the patient is HIV positive.

CULTURES

Exudative pleural effusion should be sent for culture for bacteria, fungi and mycobacteria as per the clinical suspicion. Positive culture results are obtained in less than 60% of samples. Cultures from chest tubes yield false culture results when compared with direct aspirates.

ROLE OF POLYMERASE CHAIN REACTION AND NUCLEIC ACID AMPLIFICATION TEST

Their role in diagnosing tuberculous pleural effusion is yet to be defined clearly. As per the study done by Anushree Chakraborty and et al[34] on the role of genexpert in the diagnosis of tubercular pleural effusion in India, it was concluded that pleural fluid CBNAAT, owing to its low sensitivity, should not be included in the diagnostic protocol of Tuberculous pleural effusion in high prevalence areas. A high ADA ≥ 40 U/L in combination with Light's criteria to define exudates, with lymphocyte predominance is sufficient evidence to diagnose TPE and initiate anti-tubercular therapy, thereby deferring the need to perform an invasive pleural biopsy.

PARAPNEUMONIC EFFUSION AND EMPYEMA

A parapneumonic effusion is accumulation of exudative pleural fluid associated with an ipsilateral lung infection, mainly pneumonia (mostly bacterial infections). Parapneumonic pleural effusions are classified into: Uncomplicated parapneumonic effusions, which are exudative, neutrophilic effusion. Gram stain and culture are negative, glucose level greater than 60 mg/dl and pH above 7.20. Complicated parapneumonic effusions occur due to bacterial introduction into the pleura. These have decreased glucose level, pleural fluid pH is below 7.20 and cultures are positive. It needs drainage and hence the term complicated parapneumonic effusion. Empyema is frank pus in the pleural space.

TUBERCULOUS PLEURITIS/ PLEURAL EFFUSION

A diagnosis of TPE was classified as confirmed when tuberculosis bacilli were isolated from sputum, pleural fluid or pleural biopsy, or if granulomas were observed in the latter. The diagnosis was classified as probable in patients who met the following conditions: (a) clinical picture consistent with Tuberculous pleural effusion; (b) pleural exudate with ADA levels ≥40 U/l; (c) resolution of pleural effusion with TB treatment. A positive microbiological result was defined as tuberculosis bacilli identified in a sample of PF or sputum by microscopy, culture, or PCR. A study was conducted by Rahul Gupta and et al (2018) in Jammu and Kashmir named Spectrum of pleural effusion etiology revisited in the 18–70 years of age group: A tertiary care center-based study of 1000 patients. It was found that out of a total 1000 patients, 69.5% had tuberculosis followed by malignancy (16%) with the systemic causes forming about 15% of the patients with pleural effusion. It was found more in males, associated with smoking, and the majority of patients had unilateral effusion. Eighty-nine percent of patients had exudative effusion.[35]

UNDIAGNOSED PLEURAL EFFUSION AFTER INITIAL THORACOCENTESIS

In undiagnosed pleural effusions, if all of the six following clinical parameters are met patient have a benign course, and no further evaluation is necessary: Patient are clinically stable, no weight loss, purified protein derivative test, used in detecting tuberculous pleural effusion is negative and the pleural adenosine deaminase (ADA) value is less than 43 U/mL, afebrile, pleural fluid has less than 95% lymphocytes, effusion occupies less than 50% of the hemithorax.

OBSERVATION

Approximately 15% of pleural effusion will remain undiagnosed despite extensive workup. Probably this is the best option if the patient is improving and there are no lung parenchymal infiltrates. If the patient has malignancy, spontaneous improvement is unlikely to occur. If the patient

has pulmonary embolism, the diagnosis should have been made by CT angiogram, whereas if the patient has tuberculous pleuritis, one of the pleural fluid markers for tuberculosis should have been positive.

BRONCHOSCOPY

Bronchoscopy should be performed when any of the following is present - A pulmonary infiltrate on the chest radiograph or the chest CT scan, hemoptysis (suggestive of an endobronchial lesion or pulmonary embolism), and massive pleural effusion or if the mediastinum is shifted toward the side of the effusion (endobronchial lesion is probable).

THORACOSCOPY

Thoracoscopy is indicated in the patient with an undiagnosed pleural effusion who is not improving spontaneously and in whom there is a significant likelihood that malignancy or tuberculosis is present. An advantage of thoracoscopy in the diagnosis of pleural disease is that pleurodesis can also be performed at the time of the procedure. It should be emphasized, however, that thoracoscopy rarely establishes the diagnosis of benign disease.

PLEURAL BIOPSY

Pleural biopsy is considered when tubercular or malignant etiology is suspected. The yield is increased by taking multiple samples using CT-guided cutting-needle biopsy to detect specific abnormal areas, particularly for malignancy. Closed blind pleural biopsy is not recommended now as the yield is better with image-guided pleural biopsy or thoracoscopy guided.

MANAGEMENT AND TREATMENT

Treatment of pleural effusion is dependent on its cause. Complicated parapneumonic effusions and empyemas are treated with antibiotics and should be drained. Of the common causes for exudative pleural effusions, parapneumonic effusions have the highest diagnostic priority because even with antibiotics there are high chances for development of fibrothorax. The duration of antibiotic therapy depends on many factors,

for example, the sensitivity of the organism, extent of pulmonary parenchymal and pleural disease, response to initial therapy and adequacy of drainage.[9] For malignant effusions, it signifies an advanced and incurable disease with high morbidity and dismal mean survival of less than one year stage of disease. Treatment is to palliate symptoms. Repeated thoracentesis, pleurodesis, or placement of indwelling tunneled catheters may be required for symptom palliation. Pleural effusions related to connective tissue disorders like rheumatoid arthritis and systemic lupus erythematosus are treated with steroids and resolution occurs approximately within two weeks of initiation of treatment.[10] Tubercular pleural effusions are treated with antitubercular therapy and steroids are also given in some patients. Patients with chylothorax require nutritional support with a diet rich in low-fat, medium-chain triglycerides which are absorbed directly into the portal circulation to reverse hypovolemia, immunosuppression, and protein and electrolyte deficiencies.[10] If there is a large hemothorax, tube thoracostomy is advised. repeated blood transfusion may be required. If there is retained clot, surgical evacuation should be done. Low molecular weight heparin is given in patients with pleural effusion due to pulmonary emboli. Therapeutic thoracentesis is indicated whenever a patient has developed respiratory distress. Pleurodesis (insertion of a chest tube and instillation of sclerosing agents into the pleural cavity leading to production of adhesions between the outer surface of the lung and inner surface of the chest wall, hence prevent accumulation of fluid or air in the pleural space) is indicated when there is recurrent pleural effusion. Either intercostal drainage or pigtail catheter insertion can be done. Decortication, pleurectomy, pleuropneumonectomy, closure of bronchopleural fistula with or without grafting, window operation, fenestration surgery, thoracostomy, and thoracoplasty are the various surgical modalities available.[10]

MATERIALS AND METHODS

This study was carried out in Dayanand Medical College and Hospital, Ludhiana.100 admitted patients between January 2020 to September 2021 who met the Light's criteria of exudative pleural effusion were included and followed till 3 months.

SOURCE OF DATA

The study was carried out in-hospital patients in Dayanand medical college and hospital.100 patients with exudative pleural effusion admitted in DMCH were enrolled in this study. Diagnostic/therapeutic guided thoracentesis was done. Patients who had exudative pleural effusion were included in this study. They were followed at 1 month and 3 month of discharge. Their demographic, etiological, clinical, radiological profile and outcome data were recorded and analyzed.

STUDY POPULATION

A prospective observation study was conducted in patients aged more than 18 years hospitalized with exudative pleural effusion in DMCH, Ludhiana. 100 patients admitted between January 2020 to september 2021 were included and followed till 3 months.

METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA

- age>18 years,
- Aspirable pleural effusion
- Meets the light's criteria for exudative fluid

EXCLUSION CRITERIA

- age<18 years,
- non aspirable pleural effusion,
- transudative pleural effusion
- Consent not given by the patient

INVESTIGATIONS AND INTERVENTION/SURGERY

Basic investigations like CBC, LFT, RFT, chest X ray, serum LDH (lactate dehydrogenase), ESR, ultrasound chest were done in all patients. Pleural fluid analysis was done which included – fluid glucose, protein,LDH, hematocrit fluid, triglyceride, cytological examination, ADA levels, line probe assay/CBNAAT, gram stain, culture and fluid for malignant cytology. Sputum analysis was done wherever possible. Immunological test- RA factor, thyroid stimulating hormone(TSH), antinuclear antibodies by immunofluorescence (ANA IF)/profile, complement levels, tumor markers, amoebic serology, covid RTPCR wherever indicated. Radiological tests like CECT/HRCT chest or CTPA chest were done wherever indicated.

Following interventions were required - usg guided pleural tap, PCD/ICD insertion, Streptokinase injection wherever clinically indicated, fine needle aspiration cytology and biopsy (usg/CT guided), bronchoscopy, VATS wherever clinically indicated.

TEST METHODOLOGY

LIGHT'S CRITERIA OF EXUDATIVE PLEURAL EFFUSION

Pleural fluid protein divided by serum protein greater than 0.5. Pleural fluid LDH divided by serum LDH greater than 0.6. 3. Pleural fluid LDH greater than two thirds of the upper limit of normal serum LDH.[3]

PLEURAL FLUID GRADING ON CHEST X RAY

Basis of **diagnosing and grading of pleural effusion**[36] **on chest x-ray** (CXR) as mild, moderate and massive were -

(1) Mild – only costophrenic angles are blunted.

(2) Moderate- Blunting of costophrenic angle and positive meniscus sign seen and fluid level below second intercostal space.

(3) Massive- Blunting of costophrenic angles, when fluid level is above the second intercostal space, complete opacification of hemithorax, mediastinal shift.

LIGHT'S CLASSIFICATION OF PARAPNEUMONIC AND EMPYEMA-[37]

1. **NON SIGNIFICANT PARAPNEUMONIC EFFUSION**- These patients have free-flowing fluid that is less than 10 mm thick on the decubitus chest radiograph.

2. **TYPICAL PARAPNEUMONIC EFFUSION**- In addition to above these patients have pleural fluid glucose above 40 mg/dL,pH above 7.20, LDH is below three times the upper limit of normal for serum, and the bacterial smears and cultures are negative.

3. **BORDERLINE COMPLICATED PARAPNEUMONIC EFFUSION**- These patients have negative bacterial smears and cultures and a glucose level above 40 mg/dL, pH between 7.00 and 7.20, the LDH above 3 times the upper limit of normal, or the pleural fluid is loculated.

4. **SIMPLE COMPLICATED PARAPNEUMONIC EFFUSION**- Pleural fluid pH less than 7.00, a pleural fluid glucose level less than 40 mg/dL, or a positive Gram's stain or culture. The pleural fluid does not look like pus and it is not loculated.

5. **COMPLEX COMPLICATED PARAPNEUMONIC EFFUSION**- In addition to simple complicated parapneumonic effusion, the fluid is loculated.

6. **SIMPLE EMPYEMA -** Pleural fluid that is frank pus which is either frees flowing or confined to a single locule.

7. **COMPLEX EMPYEMA**- Pleural effusion is frank pus that is multiloculated.

DIAGNOSING A TUBERCULAR PLEURAL EFFUSION

Patients were diagnosed with **"Confirmed tubercular pleural effusion"**[40] if one of the following criteria was met: **1)** AFB smear positive when pleural fluid was used as the source specimen, and the tuberculosis bacterium was grown in the culture medium, or the tuberculosis bacterium was detected by polymerase chain reaction (PCR); **2)** In the absence of granulomatous lung disease for other reasons, a pleural biopsy revealed a

granulomatous lesion with or without caseous necrosis; and **3)** The sputum culture was positive for tuberculosis bacterium and the pleural effusion improved after anti-tuberculosis treatment. "**Probable tubercular pleural effusion"** is defined as lymphocyte-predominant exudates with ADA >40 U/L in pleural fluid in the absence of evidence of malignancy and improves after anti-tuberculosis treatment.

TUBERCULAR EMPYEMA

Tuberculous empyema is a rare entity characterized by purulent pleural fluid that is loaded with tuberculous organisms on AFB stains. It is further classified as definitive and probable tubercular empyema.Tubercular empyema was defined as cases of thoracic empyema with one of the following: **1)** pleural fluid positive for Acid Fast Bacilli (AFB) smear or positive for Mycobacterium tuberculosis on CBNAAT; and/or **2)** sputum positive for AFB or positive for Mycobacterium tuberculosis on CBNAAT and having radiological lesions consistent with active parenchymal tuberculosis on chest X-Ray/CT scan of the thorax (nodular consolidation with or without cavity in apex, tree in bud appearance). Probable tubercular empyema was defined as empyema in patients who had radiological evidence of active pulmonary tuberculosis on chest X-Ray/ CT scan of the thorax or were sputum positive for AFB smear or by CBNAAT[39].

MANTOUX TEST

As per the CDC (centers for disease control and prevention) guidelines - **1)** An induration of 5 or more millimeters is considered positive in people living with HIV, a recent contact of a person with infectious TB disease, people with chest x-ray findings suggestive of previous TB disease, people with organ transplants, other immunosuppressed people (e.g., patients on prolonged therapy with corticosteroids equivalent to/greater than 15 mg per day of prednisone or those taking TNF-a antagonists). **2)** An induration of 10 or more millimeters is considered positive in people

born in countries where TB disease is common, including Mexico, the Philippines, Vietnam, India, China, Haiti, and Guatemala, or other countries with high rates of TB, people who abuse drugs, mycobacteriology laboratory workers, people who live or work in high-risk congregate settings (e.g., nursing homes, homeless shelters, or correctional facilities), people with certain medical conditions that place them at high risk for TB (e.g Silicosis, diabetes mellitus, severe kidney disease, certain types of cancer, and certain intestinal conditions), people with a low body weight (<90% of ideal body weight). **3)** An induration of 15 or more millimeters is considered positive in people with no known risk factors for TB.

DIAGNOSING MALIGNANT PLEURAL EFFUSION

Malignant effusions"[40] were also divided into confirmed and probable categories**. "Confirmed malignant effusion"** indicated positive pleural fluid cytology or positive pleural tissue histology or confirmed malignancy at another site with radiological evidence of metastatic thoracic involvement. **"Probable malignant effusion"** indicated a documented clinical diagnosis based on a suggestive radiological and clinical course and the absence of an alternative cause.

DIAGNOSING PLEURAL EFFUSION DUE TO PULMONARY EMBOLISM

Patients having exudative pleural effusion and pulmonary embolism on CT pulmonary angiogram.

DIAGNOSING HEMOTHORAX

The pleural fluid hematocrit is equal to or greater than 50% of the peripheral blood hematocrit. If a measured hematocrit is not available, a rough estimate of the hematocrit obtained by dividing the pleural fluid red blood cell (RBC) count by 100,000.

DIAGNOSING PARASITIC PLEURAL EFFUSION

If there is **1)** The pleural involvement and the feature of pleural effusion.[41] **2)** Immunoserology test result for a parasite-specific antibody, and/or on the detection of characteristic parasite eggs (in the pleural effusion, sputum, bronchial washing fluid, lung biopsy specimens or stool).[42] Improvement with treatment on clinical follow up.

DIAGNOSING PLEURAL EFFUSION DUE TO VIRAL/COVID ETIOLOGY- 1) Patients who developed pleural effusion and meets light's criteria for exudative effusion. **2)** Covid RTPCR was positive. **3)** CT chest suggestive of ground glass opacities typical of covid with CORAD 5 or 6 on CT chest. No pulmonary embolism or any evidence of myocarditis. 4) No alternative diagnosis on evaluation.

DIAGNOSING A CHYLOTHORAX

1) If pleural fluid is distinctive white, odorless, milky in appearance and milkiness or turbidity persists after centrifugation. **2)** Pleural fluid triglyceride level is above 110 mg/dL or demonstration of chylomicrons.

OUTCOME

The outcome studied were variables like status at the time of discharge-resolution/death / discharge against medical advice (DAMA), Chest X ray and any complications. Similarly at 1 month and 3 month study variables were symptomatic resolution, death/loss to follow up, follow up chest x-ray, CT chest, complications related to treatment or other, change in diagnosis and any change in treatment on follow up.

Socioeconomic status was calculated by modified kuppuswamy scale 2021

BMI-body mass index calculated as weight in kg divided by square of height in metric and classified as asia pacific classification.[43]

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables

between the study groups will be done using Student t-test and Mann Whitney U or independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and an exact test was used when the expected frequency is less than 5. A probability value (*p* value) less than 0.05 will be considered statistically significant. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

Over a period of one year and 9 months, 100 patients with exudative pleural effusion admitted in Dayanand medical college and hospital fulfilling inclusion criteria were studied.

DEMOGRAPHIC FEATURES

1. AGE DISTRIBUTION

Most of the patients in this study were middle aged adults (41-60 years) i.e. 36%, followed by young adults (20-40 years) who were 34%, 29% were old age (> 60 years) and only 1% were adolescent (< 20 years) [table1, fig 1]

2. GENDER DISTRIBUTION

As depicted in table 2 and fig 2, out of the total 100 cases studied, 27% were females and 73% were males.

3. SOCIO-ECONOMIC STATUS

On a socio-economic scale, 62% were from upper middle class and 21% from lower middle class, 11% cases from upper, 2% were from upper lower class and 4% from lower socioeconomic status. [table 3, fig 3]

4. BODY MASS INDEX

43% patients were within normal BMI range (Asian-pacific guidelines) (19-22.9 kg/m^2), 29% patients were overweight (between BMI of 23-24.9 kg/m^2), only 1% was underweight (BMI of less than 18 kg/m^2). Rest 27% were in the obese category (BMI > 25 kg/m^2) [table4, fig 4].

CLINICAL FEATURES OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION

HISTORY OF PRESENT ILLNESS

5. DURATION OF PRESENTING COMPLAINTS

Average duration of presenting complaints was 24.87 days with standard deviation of 43.14 days. 48% of patients had a history of less than 2 weeks. 42% had a history of present illness of 2-6 weeks and the rest 11% had more than 2 weeks history. The average duration of hospitalization

was found to be 12.69 days with standard deviation of 10.08 days.(table 5, fig 5)

6. PRESENTING COMPLAINTS

The most common symptom at presentation was shortness of breath which was observed in 76% of the study population, followed by complaint of fever which was present in 59% of the total cases. 53% of the patients complained of cough and only 18% had expectoration. Chest pain was a presenting complaint in 49% of the cases. History of trauma was present in 10% of the cases and 4% had hemoptysis. Other complaints like weight loss (10%), loss of appetite (4%), and pain abdomen (1%) were present in 15% of the cases. [table 6, fig 6]

7. PAST HISTORY

Out of the 100 patients, 22% had diabetes mellitus and hypertension each, 10% had a history of tuberculosis in the past, 6% had CLD and 2% had CKD. 4% patients had previous history of malignancy (2% CA lung, 1% CA ovary and 1% Hodgkin's lymphoma). History of coronary artery disease was present in 2% and hypothyroidism in 1% of the cases. 3% of the patients were HCV reactive, 1% had history of HBV and another 1% had HIV positive status.1% patient had covid positive history, 1% had history of hysterectomy,1% had bronchial asthma and 1% had history of ventilatory support in the recent past. [table7, fig 7]

8. SUBSTANCE ADDICTION

27% of the patients had a history of addiction. 17% were addicted to alcohol, 8% to smoking, 1% to tobacco and another 1% were addicted to opioids. [table 8, fig 8]

9. GENERAL PHYSICAL EXAMINATION

35% patients needed oxygen support who had saturation less than 94% at room air. Icterus, lymphadenopathy and clubbing were present in 3% each. [table 9, fig 9]

10. CHEST EXAMINATION

On chest examination, the most common findings were asymmetrical chest (81%), tracheal deviation (70%), decreased tactile fremitus (100%), dullness to percussion (100%). On auscultation, air entry was absent in certain areas on the right side in 51 %, at the left side in 42% and bilaterally in 7% according to the amount of pleural fluid [table 10, fig 10].

11. OTHER SYSTEMIC EXAMINATION

Other systemic findings like as cites were present in 7%, encephalopathy was present in another 7% and 1% had muffled heart sounds on auscultation. Right upper quadrant tenderness was present in 1% of the cases. 1% of patients had choroid tubercle on fundus examination [table 11, fig 11].

RADIOLOGICAL TESTS

12. CHEST X RAYS

Chest x-ray was done in all the patients. Right sided effusion was present in 51%, 42% had left sided effusion and the rest 7% had bilateral effusion. 76% had mediastinal shift, 21% had opacity in the lung field. Other findings like cavity (3%), fibrosis (3%), ground glass opacities (1%), miliary mottling (1%), pneumothorax (1%), and rib fracture (1%) were also observed [table 12&13, fig 12&13].

16. ULTRASONOGRAPHY CHEST

Ultrasonography chest was done in all the patients. As observed on chest x-rays, on USG chest also right sided effusion was present in 51%, 42% had left sided effusion and the rest 7% had bilateral effusion [Table 14, Fig 14].

On the ultrasound, effusion was graded as mild, moderate and significant depending on the amount of pleural fluid. Most of the patients had moderate effusion i.e. 59%, 21% had mild and the rest 20% fell into the significant category. Out of these, 13% patients had loculated pleural effusion (single locule). Another 17% had multiloculated or the septated effusion [Table 15, Fig 15].

18. COMPUTED TOMOGRAPHY CHEST

CT chest was done in 27 patients as per the clinical indication. 48.14% cases were having left sided effusion, 40.07% were having right sided effusion and the rest 11.11% were having bilateral effusion. Of these 59.25% had moderate effusion, 29.62% had significant effusion and 11.11% had mild effusion. CA lung was the most common finding in 14.81%, followed by pulmonary embolism present in 11.11% of patients. Empyema on CT chest was present in another 11.11% of patients while ground glass opacities with CORAD 6 was present in 7.4% of patients. Lung mass was present in 7.4% of patients. Other findings were disseminated kochs, cavity, consolidation, necrotizing lymphadenopathy and hydropneumothorax (3.7% each) [table 16, fig 16].

19. USG GUIDED THORACOCENTESIS

USG guided diagnostic pleural tap was done in all the patients. 28% underwent therapeutic tap as well. Following pleural tap, 4% patients developed procedure related complications - hydropneumothorax in 3% and hemothorax in 1% [table 17 & 18, fig 17, 18 & 19].

PLEURAL FLUID ANALYSIS

20. APPEARANCE/ COLOR OF PLEURAL FLUID

On pleural fluid examination at first gross appearance of the fluid was noted. Most of the patients i.e. 64% had straw colored aspirate, 22% had pus, 11% had hemorrhagic fluid, 2% had anchovy sauce appearance and 1% had milky/chylous effusion [table19, fig 20].

21. ETIOLOGICAL CLASSIFICATION

All these patients met Light's criteria for exudative pleural effusion. They were further classified according to their underlying etiology. Out of the 100 cases studied, tuberculosis was found to be the most common cause accounting for 42% of the cases. They were further subdivided into probable tubercular effusion (28%), confirmed tubercular effusion (8%), definitive tubercular empyema (4%) and probable tubercular empyema (2%). Empyema was seen in 15% and parapneumonic effusions were

present in 16% of the total cases. Empyema was subclassified into simple empyema and complex empyema and accounted for 6% and 9% respectively. Similarly, parapneumonic effusions were subclassified into typical (1%), borderline (0%), simple complicated (7%) and complex complicated (8%). Malignant etiology was present in 9% of the cases which was further divided into probable (2%) and confirmed malignant pleural effusion (7%). 11% patients had other causes like hemothorax (3%), pulmonary embolism (3%), parasitic pleural effusion (2%), viral etiology (2%), and chylothorax (1%). However 7% of the exudative pleural effusions remain undiagnosed i.e. unknown etiology (table 19 &20, fig 21&22)

TUBERCULOUS PLEURAL EFFUSION

Out of the 42 cases of tubercular pleural effusion, 35 patients had straw colored aspirate while the rest 7 had pus (table 21, fig 23). All the patients in the tubercular group had TLC less than 2500 cells/mm^3 with mean of 519.06 cells/mm^3 (table 22&23, fig 24 & 25).The effusion was lymphocytic predominant in 95.24% cases of TB and the rest 4.76% had neutrophil predominant effusion (Table24, Fig 26). Lymphocyte to neutrophil ratio was more than 0.75 in 38 cases (p value 0.0001) with sensitivity of 61.29% and specificity of 89.47%. Positive predictive value (PPV) of Lymphocyte to neutrophil ratio was 90.48% and negative predictive value (NPV) 58.62% (Table 25 & 26, Fig 27 & 28). All the patients had ADA more than 40 U/L (n-42), 40 patients had pleural fluid ADA value of more than 60 U/L and only 2 had ADA between 40-60 U/L with p value of 0.0001 which is statistically significant (table 27 & 28, fig 29 & 30). Sensitivity of ADA for diagnosing tuberculous pleural effusion was 56.76% and specificity of 100%. PPV of ADA for diagnosis of tuberculosis was 100% and NPV was 44.83% (Table 29, Fig 31). In this study, 75.5% of the patients who had both ADA > 40 U/L and lymphocyte to neutrophil ratio >0.75 were found to have tubercular pleural effusion [Table 30, Fig 32]. In diagnosing tuberculous effusion the combined sensitivity was 74.51% and

specificity was 91.84% (table 32, fig 33). Mean pleural fluid glucose was found to be 87.31mg/dl. 30 patients were having pleural fluid glucose levels between 40-100mg/dl (p value-0.0001), 8 patients had more than 100 mg/dl and 4 patients had less than 40mg/dl (p value 0.0001) (table 33 & 34, fig 34). The pleural fluid protein to serum protein ratio was more than 0.5 in all the 42 cases [table 35, fig 35]. Pleural fluid LDH to serum LDH ratio was between 1-1.5 in 18 patients, 16 patients had ratio more than 1.5 and 8 patients had between 0.6-1.5 (p value - 0.375) (table 36, fig 36). CBNAAT pleural fluid was positive in 11 patients (n-42, 26.1% in its group) with sensitivity of 26.19% and specificity of 100%. Positive predictive value was 100% and negative predictive value was 65.17% (p value 0.001) [Table 38 & 39, Fig 37]. Pleural fluid AFB was positive in 2 patient's i.e 4.76% in TB group (table 42, fig 40). Histopathologically, 1 patient was found to have lymph node tuberculosis on biopsy and another 1 had chronic granulomatous inflammation [table 45, fig 42]. Other tests like sputum AFB were positive in 1 patient and 5 (11.9% in its own group) patients had a mantoux test positive [table 45, fig 42].

PARAPNEUMONIC EFFUSION AND EMPYEMA

Pleural effusions were categorized into parapneumonic pleural effusion 16% and empyema 15% [table 19, fig 21]. On gross appearance all 15 empyema were pus and parapneumonic effusions were straw colored (table 21, fig 23). 86.67% of empyema and 87.50% of parapneumonic effusions were neutrophil predominant with average pleural fluid TLC as 1238.75 cell/mm^3 with standard deviation of 1073.057 cells//mm^3 in parapneumonic effusion and 1911.33 cell/mm^3 with standard deviation of 2570.444 cell/mm^3 in empyema [table 22 & 24, fig 24 & 26] (p value 0.017). Only 2 patients with empyema had more than 2500-5000 cells/mm^3 TLC and 1 patient with more than 5000 cells/mm^3 TLC in pleural fluid, the rest 12 had less than 2500 cells/mm^3 (P value 0.09) [table 23, fig 25]. Similarly in parapneumonic effusion 3 patients had TLC between 2500-500 cells/mm^3 and rest were having less than 2500 cells/mm^3 [table

23, fig 25] (P value 0.09). Pleural fluid lymphocyte to neutrophil ratio was less than 0.75 in 13 cases each in empyema group and parapneumonic effusion (p value-0.0001) [table 25, fig 27]. Mean pleural fluid glucose in empyema was 28.13 mg/dl with standard deviation of 22.853 mg/dl, parapneumonic effusions had glucose of 36.87 mg/dl with standard deviation of 45.006 mg/dl (p value-0.0001) [table 33]. Majority of the patients in both empyema (n-15, 10 patients i.e. 66.6%) and parapneumonic (n-16, 12 patients i.e. 75%) groups had pleural fluid glucose less than 40mg/dl. 4 patients in the empyema group i.e. 26.66% and 4 patients in parapneumonic group i.e. 25% had pleural fluid glucose between 40-100mg/dl. And 6.66% i.e 1 patient in the empyema group had glucose >100 mg/dl [table 34, fig 34] (p value-0.0001). In empyema plural fluid protein to serum protein ratio was more than 0.7 in 8 patients while rest 7 had less than 0.7. in parapneumonic group this ratio was less than 0.5 in 2 patients and rest had more than 0.5 (p value- 0.313) [table 35 fig 35]. In case of empyema (n-15) pleural fluid LDH to serum LDH ratio was more than 1 in 14 cases and in parapneumonic effusions (n-16), all patients had high serum LDH to pleural fluid LDH ratio i.e. more than 1 [table 36, fig 36]. ADA was found to be more than 40 U/L in 8 cases of empyema (53.3%) and 12 patients of parapneumonic effusion (75%) [table 27, fig 29]. However, only 13.3% cases of empyema (2, n-15) and 6.25% cases of parapneumonic effusion (1, n-16) had both ADA >40 U/L and lymphocyte to neutrophil ratio of more than 0.75 (table 30, fig 32). Overall, Gram's stain was positive in 5% of the total patients, 2% had Gram positive cocci, 2% had Gram negative cocci and 1 % had Gram negative bacilli [table 40, fig 38]. Pleural fluid cultures were positive in 21% of the cases, most common organism isolated was MRSA i.e. methicillin resistant Staph aureus accounting for the 8% of the total cases studied, followed by E.coli (4%), K.pneumoniae (3%), P.aeruginosa (2%) and Acinetobacter (2%). 1% had growth of Burkholderia and another 1% had polymicrobial growth (both Streptococcus pneumoniae and Pseudomonas

aeruginosa were grown in culture) [table 41, fig 39]. Other tests like sputum culture were positive in 2 cases, 1 had growth of Escherichia coli and other had Klebsiella pneumoniae [table 45, fig 42].

MALIGNANT PLEURAL EFFUSION

Malignant pleural effusion constituted 9% of the total cases. Out of these, 77.77% (7 cases) had confirmed malignant pleural effusion (n-9) and 22.3% (2 cases) had probable malignant pleural effusion (table 20, fig 22). Of these 33.33% (3 patients) had hemorrhagic pleural effusion on gross appearance and the rest 66.66% (6 patients) had straw colored effusion [table 21, fig 23] (p value-). 8 patients had pleural fluid TLC count less than 2500 cells/mm^3 and only 1 had pleural fluid TLC more than 2500 cells/mm^3 (table 23, fig 25) with mean TLC of 588.89 cells/mm^3 (table 22, fig 24). 88.89% had predominantly lymphocytic pleural effusion and 11.11% had neutrophil predominant effusion (table 24, fig 26). Lymphocyte to neutrophil ratio was found to be more than 0.75 in 7 patients (77.77%) of malignant pleural effusion [table 25, fig 27] (p value 0.0001). ADA of less than 40 U/L was found to be present in (44.4%) 4 patients while 5 (55.6%) patients had ADA more than 40 U/L [table 28, fig 30] (p value- 0.186). Of the total 51 cases who had combined ADA >40 U/L and lymphocyte to neutrophil ratio >0.75, only 10.2% were malignant [table 30, fig 32]. Average pleural fluid glucose was found to be 110.22 mg/dl (table 33). All patients had pleural fluid glucose more than 40 mg/dl [table 34, fig 34]. All patients had pleural fluid protein to serum protein ratio of more than 0.5 [table 35, fig 35]. 5 patients had pleural fluid LDH to serum LDH ratio more than 1.5, rest 4 had this ratio between 0.6-1 (p value 0.375) (table 36, fig 36). Fluid for malignant cytology was positive in 7 patient's i.e 77.77% in malignant pleural effusion group [table 42, fig 40]. Fluid for malignant cytology has sensitivity of 77.78% with 100% specificity [table 43 & 44, fig 41] (p value 0.001). Tumor markers were positive in 22.22% of the malignant cases (table 45, fig 42). On CT chest 4 patients had CA lung (table 16, fig 16). On histopathology 4 patients (n-9, 44.44%)

had CA lung- adenocarcinoma while 1 patient (n-9, 11.11%) had CA lung-non small cell carcinoma, one had unknown primary [table 45, fig 42]. Of the 9 patients with malignant pleural effusion, 2 patients had previous history of CA lung, 1 had previous history of CA ovary, [table 7, fig 7].

OTHER ETIOLOGIES

11% patients had been grouped under the heading of 'other' causes which includes pulmonary embolism (3%), hemothorax (3%), parasitic (2%), viral (2%) and chylothorax (1%) [table 20, fig 22]. In pulmonary embolism, 2 patients had hemorrhagic pleural fluid while 1 had straw colored appearance; all 3 cases diagnosed with hemothorax were hemorrhagic in appearance; 2 parasitic effusion had anchovy sauce appearance; both the viral pleural effusion were straw colored and chylothorax had milky/chylous appearance of pleural fluid (table 46, fig 43). On pleural fluid lab analysis it was found that mean TLC was 166.66 cell/mm^3 in pulmonary embolism with lymphocyte predominance in 2 while 1 had neutrophil predominance. Similarly in hemothorax mean TLC was 766.6 cells/mm^3 and was neutrophil predominant in two cases and one had lymphocyte predominance; parasitic had mean TLC of 805 cells/mm^3 with neutrophil predominance; viral etiology had mean TLC of 4000 cells/mm^3 (one neutrophil predominant and other lymphocyte predominant). Chylothorax had TLC of 100 cells/mm^3 and it was lymphocyte predominant (table 47, fig 44). The mean pleural fluid protein in pulmonary embolism was 3.93 g/dl, in hemothorax 4.8 g/dl, in parasitic 2.65 g/dl, in viral 2.85 g/dl and chylothorax had 2.9 g/dl of protein (table 47, fig 46). Mean LDH was found to be 286.6 U/L in pulmonary embolism, 640 U/L in hemothorax, 315 U/L in parasitic, 845.5 U/L in viral and LDH was 221 U/L (table 45, fig 46). In 3 patients with hemothorax PCV of pleural fluid was more than the 50% of blood PCV (table 49). On CT chest done on patients with pleural effusion, 3 patients were found to have pulmonary embolism and 2 patients had ground glass opacities with CORAD 6 were labeled as viral pleural effusion [table 49]. 2 patients had positive entamoeba

histolytica serology in the parasitic group [table 50]. 2 patients had covid RTPCR positive - viral pleural effusion,1 patient had triglyceride of 240 mg/dl and was classified as chylothorax [table 50].

UNKNOWN ETIOLOGY

Rest 7% of the patients remain undiagnosed. Of these 3 had hemorrhagic pleural effusion while 4 had straw colored [table 21, fig 23]. Average TLC count was 300 cells/mm^3 with standard deviation of 300 cells/mm^3 (p value 0.017) [table 22, fig 24]. All the patients had TLC of less than 2500 cell/mm^3 (table 23, fig 25). Three had lymphocytic predominant effusion while four had neutrophil predominant effusion (table 24, fig 26). All patients had ADA of less than 40 U/L except one who had value between 40-60 U/L (table 27, fig 29). 5 patients had pleural fluid protein to serum pleural fluid ratio between 0.5-0.7, rest 2 had more than 0.7 (table 35, fig 35). Average glucose was 87.57 mg/dl (table 33). Pleural fluid LDH to serum LDH ratio was more than 1 in all the 7 patients [table 36, fig 36].

OTHER BLOOD INVESTIGATIONS

On routine lab investigations, 37% were having hemoglobin more than 12 g/dl, 60% had mild to moderate anemia at presentation and 3% had severe anemia (table 54, fig 50). 42% had TLC of less than 10,000 10^3/UL, 44% had between 10,000 to 20,000 10^3/ul and 14% had more than 20,000 10^3/ul. ESR was more than 40 in 84%. At admission serum creatinine was raised >1.1mg/dl in 35%. Serum SGOT and SGPT were more than 40 U/L in 58% and 59% respectively (table 54, fig 50). Average Serum protein was found to be as 6.29 g/dl in tubercular group, 6.32 g/dl in malignant group, 6.23 g/dl in empyema group, 6.22 g/dl parapneumonic group, 5.62 g/dl in ‘other’ group and 6.46 g/dl unknown group (p value 0.451) (table 37). Average serum LDH was found to be 222.05 U/L in tubercular group, 287 U/L in malignant group, 263.57 U/L in empyema group, 214.31 U/L parapneumonic group, 299 u/l in ‘other’ group and 152.43 U/L unknown group (p value 0.092) [table 37].

TREATMENT

All these patients were treated according to the etiology and some were empirically started on antibiotics. Of the 100 patients studied, 64% received antibiotics, 42% were put on ATT (1 patient on modified ATT), 7% patients were started on chemotherapy, 3% patients underwent surgical procedure in the form of VATS [table 55, fig 51]. 5% patients were given steroids and another 2% received both steroids and antivirals. 3% patients received LMWH. 5% patients received inotropic support in view of hemodynamic instability [table 56, fig 53].

DRAINAGE AND FIBRINOLYTIC THERAPY

Drainage via percutaneous catheter or intercostal tube was performed in 50% of the cases and 16% received fibrinolytic agent- streptokinase. As per the etiology, in tuberculous pleural effusion (n-42), 9 patients (21.4%) needed drainage and of these 3 patients (7.14%) required streptokinase also. In empyema (n-15) all 15 patients required drainage (100%), and 8 patients required streptokinase injection (53.3%). In parapneumonic effusions (n-16), 15 patients (93.75%) required drainage and of these 5 patients (31.25%) required streptokinase injection (n-16). In malignant pleural effusion (n-9), three patients i.e. 33.33% required drainage. Seven (20%) patients with other causes (n-10) required drainage (three hemothorax, two parasitic,one pulmonary embolism and one chylous effusion) while only one patient with unknown etiology (n-7) required drainage via percutaneous catheter [table 51 & 58, fig 47 & 53].

OUTCOME

Outcome of the patients was observed at discharge, at 1 month and at 3 month of discharge. At the time of discharge, 3 patients of the total 100 (3%) went DAMA - 2 were malignant pleural effusion cases and one was parapneumonic effusion. 7 patients (7%) had expired at discharge - 3 were empyema, 2 were parapneumonic, 1 tuberculous and 1 from other causes (hemothorax). At 1 month, 4 more patients expired taking the total expiry 11 out of 100 i.e 11%. 2 patients who expired at 1 month were

having malignant pleural effusion, 1 had empyema and the other 1 had parapneumonic effusion. 1 patient from malignant pleural effusion was lost to follow up. At 3 month period, 2 more patients expired taking total count to 13 i.e. overall mortality at 3 month follow up of patients with exudative pleural effusion due to various causes was 13%.1 patient from malignant pleural effusion group was lost to follow up at 3 month period [table 52 & 59, fig 48 & 55].

Chest x ray was done at discharge, 1month and 3 month period. Of the 92 patients left (7 expired and 1 went DAMA), pleural effusion resolved completely in 6 patients (6.5%). In case of tuberculous pleural effusion, (n-42) at the time of discharge, 1 patient (2.3%) had complete resolution on chest x ray, 21 patients had mild pleural effusion (50%), 13 patients (30.95%) had moderate pleural effusion, 6 patients (14.2%) were seen with PCD in situ on chest x ray and discharged. At 1 month 18 (42.85%) patients had complete resolution while another 18 (42.85%) had mild pleural effusion. 1 patient (2.38%) had recurrence. At 3 months, 27 patients (64.28%) had complete resolution, 7 patients (16.66%) developed pleural thickening and 1 patient (2.38%) was having recurrence on chest x-ray [table 60, fig 56].

In malignant pleural effusions (N-9) at the time of the discharge one patient (11.11%) had persistent significant pleural effusion, four had moderate (44.44%) and three had (33.33%) mild pleural effusion. At 1 month, four (44.22%) patients had mild and two patients (22.22%) had moderate pleural effusion. At 3 months mild pleural effusion was present in two patients (22.22%) while other two (22.22%) had recurrence, and for the rest five data were not available (lost to follow up or expired) [table 60, fig 56].

Similarly in empyema (n-15) and parapneumonic (n-16) effusion, four patients (26.6%) and six patients (37.5%) respectively had mild degrees of pleural effusion respectively at the time of discharge. In the empyema group three (20%) patients had complete resolution while three patients

(20%) were discharged with PCD in situ. Moderate effusion was seen in two cases (13.3%). In parapneumonic effusions also, one patient (6.25%) had complete resolution, four patients (25%) were discharged with PCD in situ and three (18.75%) had moderate effusion. At 1 month in the empyema group eight (53.3%) had complete resolution, three (20%) had mild pleural effusion and one (6.66%) had moderate pleural effusion. In the parapneumonic group, five had (31.25%) complete resolution, six had mild (37.5%), one patient (6.25%) had moderate effusion while one patient (6.25%) developed pleural thickening on chest x-ray. At 3 months, ten patients (66.6 %) in the empyema group had complete resolution, one patient (6.66%) had mild pleural effusion, one patient (6.66%) developed pleural thickening. In the parapneumonic group at 3 month, six patients (37.5%) had complete resolution, four (25%) had mild, one (6.25%) moderate and one patient (6.25%) developed pleural thickening [table 60, fig 56].

In ‘other’ causes, at discharge mild effusion was present in 9 cases and one patient had resolution. At 1 month, six patients had complete resolution (three pulmonary embolism, two parasitic and one hemothorax) and three had mild pleural effusion (two viral, one chylous), one patient developed pleural thickening (hemothorax). At 3 months, five patients had complete resolution (three pulmonary embolism, two parasitic,), two patients had mild (viral, chylous), two patients developed pleural thickening (hemothorax) at 3 month period (table 53 & 60, fig 50 & 57). Patients with unknown etiology six patients were mild while one patient had moderate pleural effusion. At 1 month follow up, three patients had recurrence, one patient had complete resolution, one had mild pleural effusion other one had moderate. One patient developed pleural thickening. At 3 month follow up, one more patient developed pleural thickening [table 60, fig 56].

On follow up CT chest done at 1 month, there were new findings in the two patients, one patient who was initially being treated as tuberculous

pleural effusion was found to have anterior mediastinal mass. One patient who was initially categorized under unknown etiology was reclassified as having malignant pleural effusion and was found to have lymphangitis catcinomatosis.One patient had persistent left moderate effusion despite antibiotics and initially had growth of burkholderia (? contamination) was also reclassified under the heading of unknown etiology. At 3 months,one patient of unknown etiology had persistent moderate pleural effusion and therefore remained undiagnosed [table 61 & 62, fig 57 & 58].

At 1 month discharge, three patients (3%) had change in diagnosis as discussed above, Two patients were reclassified as malignant pleural effusion, one was being treated initially as tuberculous pleural effusion, another was having unknown etiology at the time of discharge.One patient who was being treated initially as parapneumonic effusion was reclassified under the unknown etiology in view of persistent moderate pleural effusion. At 3 months, there was no change in overall diagnosis and seven patients were still having unknown etiology [table 62, fig 59].

At the time of discharge one patient with tuberculous pleural effusion developed hydropneumothorax. At 1 month of the 42 diagnosed pleural effusions initially, one was having recurrence and after evaluation was reclassified into malignant effusion, one developed acute kidney injury, one developed chest pain, and seven patients had drug induced hepatitis. At 3 month one patient had chest pain and one had recurrence of pleural effusion in the tuberculous pleural effusion group. In patients with malignant pleural effusion one patient had disease progression and developed metastasis to the brain while one patient contracted covid pneumonitis and died of its complications. In patients with unknown etiology of pleural effusion three patients had recurrence of effusion at 1 month and two patients had recurrence at 3 month period, however remained undiagnosed [table 63, fig 59].

At 1 month of discharge, of the 42 patients who were put on ATT at discharge (1 on modified ATT), eight were receiving modified ATT, one

patient was started on chemotherapy, PCD was removed in two patients. At 3 month period all these patients continued with the treatment they were receiving at 1 month. In patients with malignant pleural effusion, two patients required therapeutic pleural tap at 3 month period. In empyema group one patient had PCD removal at 3 month period. In patients with unknown etiology, one was started on chemotherapy at 1 month, one had therapeutic pleural tap and one patient underwent VATS. At 3 months among patients with unknown etiology one patient had PCD insertion and another one had VATS [table 64 fig 60].

Total 13 patients expired till 3 month follow up. On comparing the data of non survivor group with survivor group (n 98, two lost to follow up), it was observed that the four patients were from empyema group, three patient from parapneumonic and malignant etiology each, two patients were from 'other' group and one patient from tubercular group [table 59, fig 55].The mean age of patients in expired group was 57.62 years with standard deviation of 12.67 years. Amongst expired cases 53.8% were females and the rest 46.2% were males (p value- 0.037). Shortness of breath (eleven cases), fever (ten cases), cough (eight cases), expectoration (six cases) and trauma (two cases) were the chief complaints. Past history of diabetes mellitus in six patients, hypertension four patients, CLD, bronchial asthma, CA lung and CA ovary in one patient each was present. One patient had HCV positive status. Among the expired cases, 38.5% cases had normal BMI, 30.8% were obese, 23.1% were overweight and 7.7% were underweight. Evidence of other system involvement on physical examination was as follows- two patients had icterus (p value 0.045), one patient had muffled heart sound (p value 0.133), five patients had encephalopathy (p value 0.0001) and ascites was present in three patients (p value 0.054). Of the 13 patients who expired, nine were anemic with average hemoglobin of 10.67 g/dl with standard deviation of 2.21 g/dl, (p value 0.155), average TLC of 23.33 10^3/ul with standard deviation of 10.02 10^3/ul (p value- 0.000), creatinine was more than 1.1 mg/dl in

76.9% (p value- 0.001), albumin was less than 3.5g/dl in twelve patients (p value- 0.014), SGOT and SGPT were raised in ten and eleven patients respectively, serum LDH of more than 200 U/L was present in 84.6% (p value 0.003). Seven patients had right sided effusion, four patients had left sided effusion and two had bilateral pleural effusion (p value 0.389). Eleven patients had moderate and two had significant pleural effusion. It was multiloculated or septated in five patients (p value- 0.046). Pleural fluid glucose was less than 40 g/dl in eight patients (p value- 0.003). The mean pleural fluid LDH was 932.38U/L with standard deviation of 514.03 U/L (p value 0.231) and pleural fluid protein was 4.33 mg/dl with standard deviation of 0.99 mg/dl (p value 0.609).The average pleural fluid TLC was 1460.77 cells/mm^3 with standard deviation of 1962.57 cells/mm^3(p value 0.125) [table 65 & 66].

DEMOGRAPHIC FEATURES OF STUDY POPULATION

1. AGE DISTRIBUTION

Age group (years)	No. of cases	Percentage
< 20	1	1.0%
21-40	34	34.0%
41-60	36	36.0%
> 60	29	29.0%
Total	100	100.0%

Table 1- Age (years) distribution of patients with exudative pleural effusion.

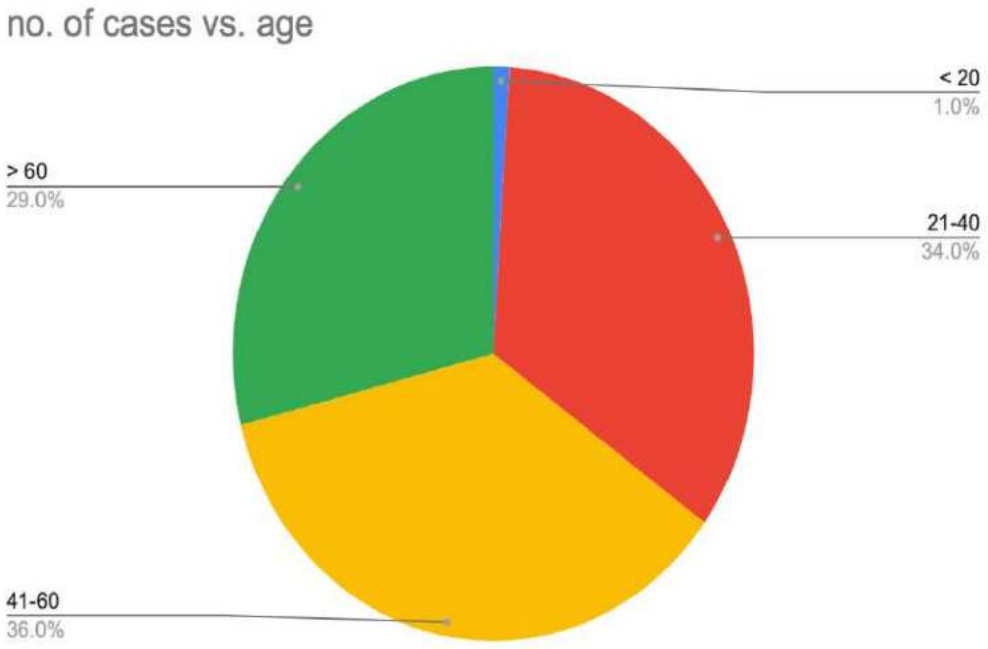

Figure 1- Age (years) distribution of patients with exudative pleural effusion.

2. GENDER DISTRIBUTION

Gender	No. of cases	Percentage
F	27	27.0%
M	73	73.0%
Total	100	100.0%

Table 2- Gender distribution of patients with exudative pleural effusion (F-Females, M-males)

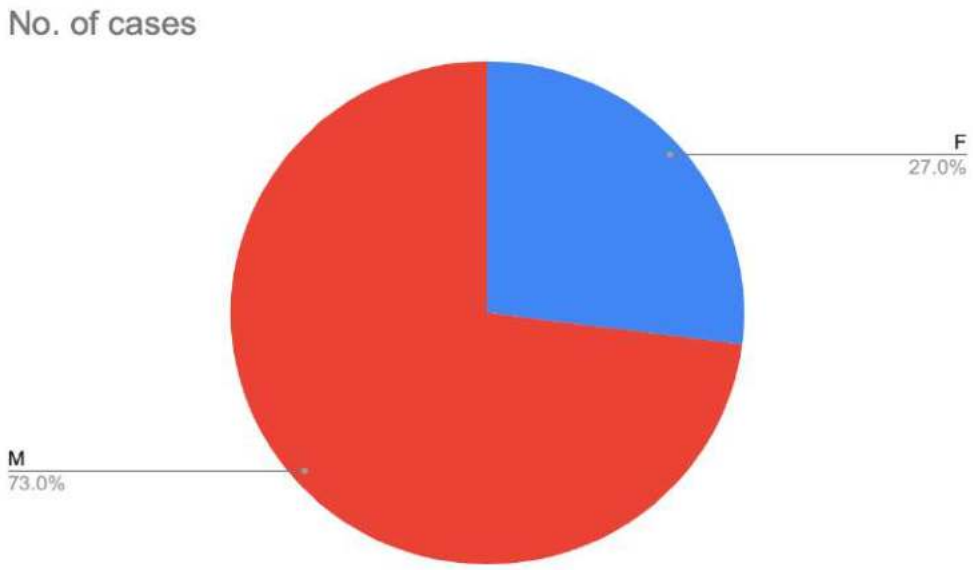

Figure 2- Gender distribution of patients with exudative pleural effusion (F-Females, M-males)

3. SOCIO-ECONOMIC STATUS

Socio-economic status	No. of cases	Percentage
ULC	2	2.0%
LMC	21	21.0%
LOWER	4	4.0%
UMC	62	62.0%
UPPER	11	11.0%
Total	100	100.0%

Table 3- Distribution of cases as per socio-economic status (UMC- Upper middle class, ULC- Upper lower class, LMC-lower middle class)

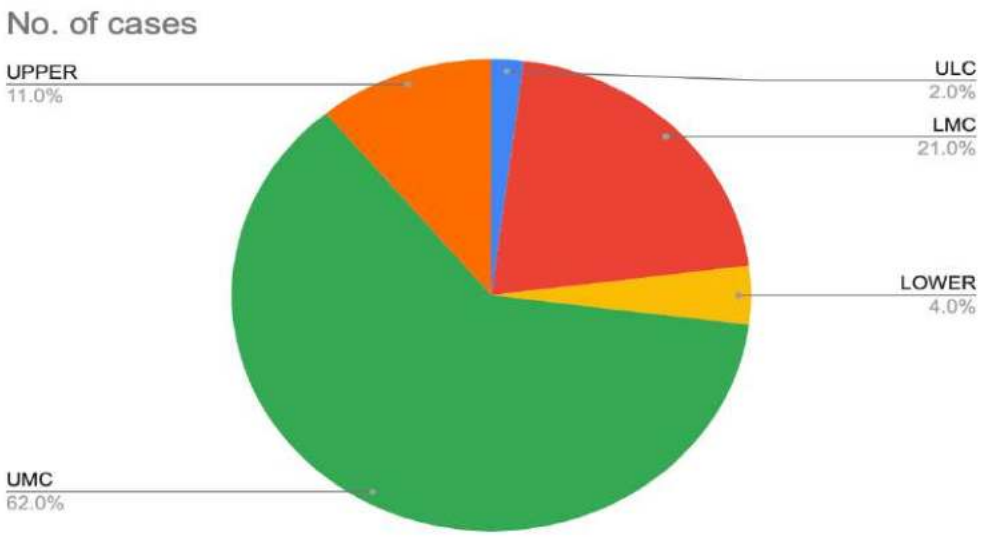

Figure 3 - Distribution of cases as per socio-economic status (UMC- Upper middle class, ULC- Upper lower class, LMC-lower middle class)

4. BODY MASS INDEX (KG/M^2)

BMI GROUP	No. of cases	Percentage
< 18.9	1	1.0%
19-22.9	43	43.0%
23-24.9	29	29.0%
> 25	27	27.0%

Table 4- Distribution of cases as per the body mass index (BMI) (Asian-pacific guidelines)

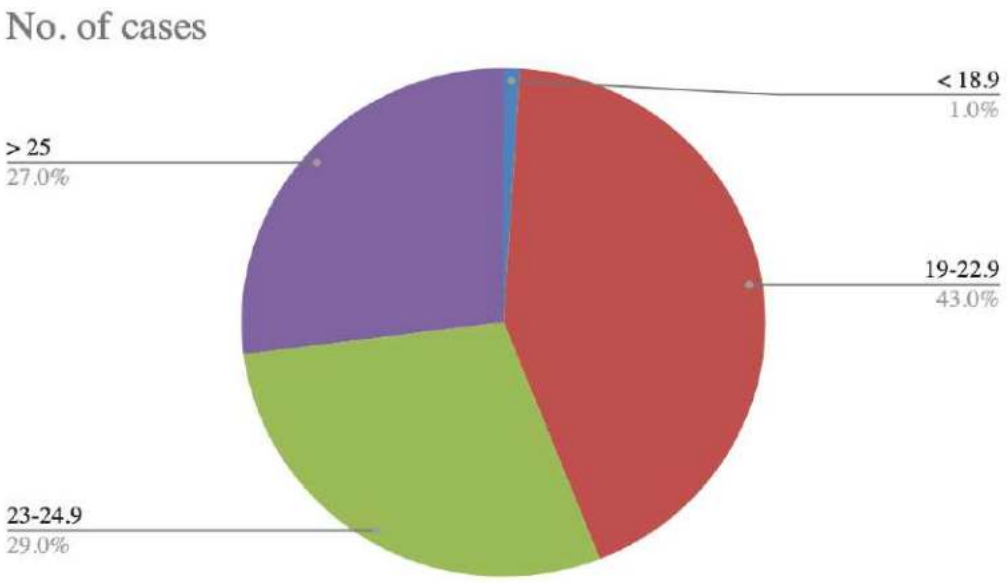

Figure-4 Distribution of cases as per the body mass index (BMI) (Asian-pacific guidelines)

CLINICAL FEATURES OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION

HISTORY OF PRESENT ILLNESS

5. DURATION OF PRESENTING COMPLAINTS

Duration of illness(days)	No. of cases	Percentage
< 14	48	48.0%
14-42	41	41.0%
> 42	11	11.0%
Total	100	100.0%

Table 5- Duration of presenting complaints (duration of illness in days)

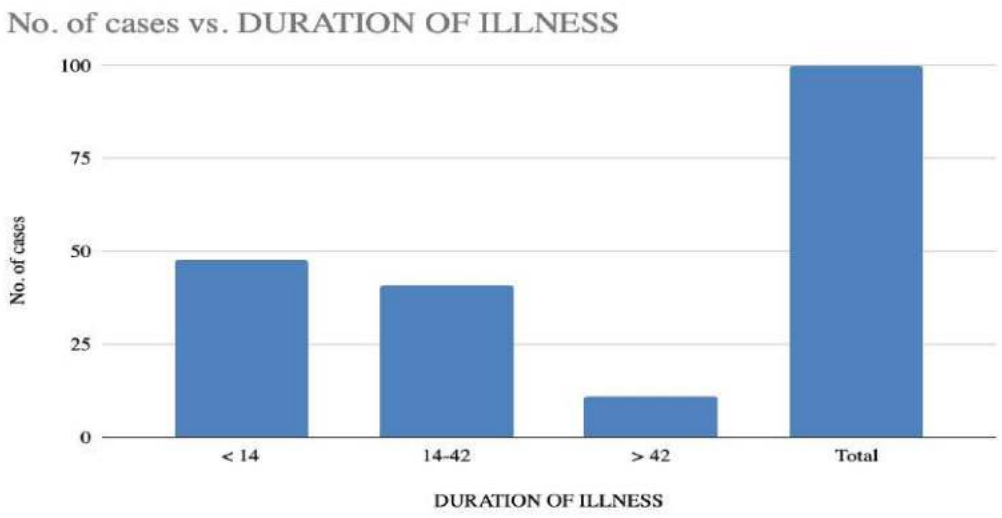

Figure 5- Duration of presenting complaints (duration of illness in days).

6. PRESENTING COMPLAINTS

Present history	No. of cases	Percentage
CHEST PAIN	49	49.0%
SOB	76	76.0%
FEVER	59	59.0%
TRAUMA	10	10.0%
COUGH	53	53.0%
EXPECTORATION	18	18.0%
HEMOPTYSIS	4	4.0%
OTHER	15	15.0%
AP LOSS	4	4.0%
PAIN ABDOMEN	1	1.0%
WT LOSS	10	10.0%

Table 6- Distribution as per the presenting complaints (SOB- shortness of breath, AP LOSS- appettite loss, WT LOSS- weight loss)

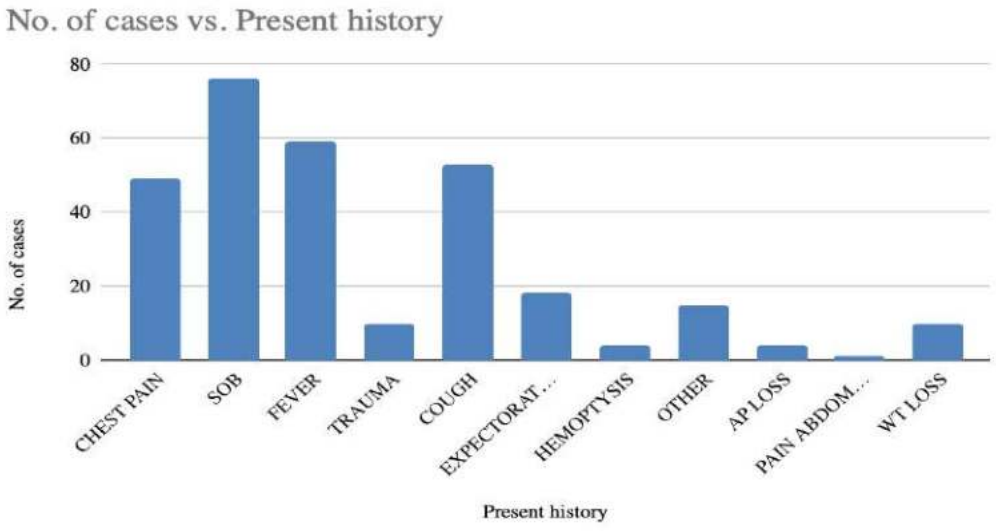

Figure 6- Distribution as per the presenting complaints (SOB- shortness of breath, AP LOSS- appetite loss, and WT LOSS- weight loss)

7. PAST HISTORY

Past history	No. of cases	Percentage
TB	10	10.0%
HTN	22	22.0%
DM	22	22.0%
CLD	6	6.0%
CKD	2	2.0%
PLHIV	1	1.0%
HCV	3	3.0%
HBV	1	1.0%
OTHER	11	11.0%
BA	1	1.0%
CA LUNG	2	2.0%
CA OVARY	1	1.0%
CAD	2	2.0%
COVID POSITIVE	1	1.0%
HL	1	1.0%
HYPOTHYROIDISM	1	1.0%
HYSTERECTOMY	1	1.0%
ONVENTILATORY SUPPORT	1	1.0%

Table 7- Distribution as per the past history (TB- tuberculosis, HTN- hypertension, DM- diabetes mellitus, CLD- chronic liver disease, CKD, chronic kidney disease, PLHIV- people living with HIV, HCV- hepatitis c virus, HBV- hepatitis b virus, CA-carcinoma, BA- bronchial asthma, CAD- coronary artery disease, HL-hodgkin's lymphoma)

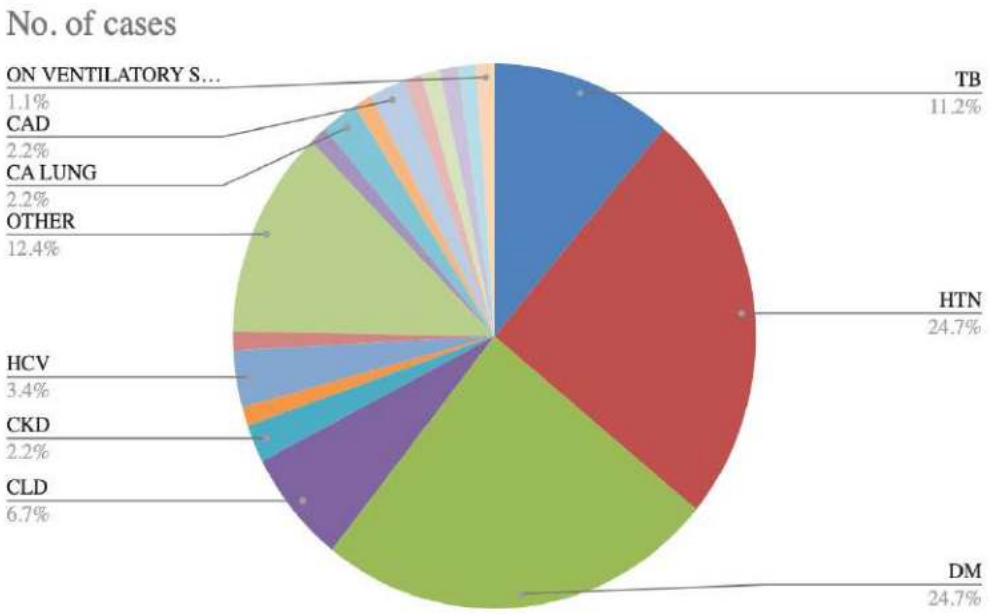

Figure 7- Distribution as per the past history (TB- tuberculosis, HTN- hypertension, DM- diabetes mellitus, CLD- chronic liver disease, CKD, chronic kidney disease, PLHIV- people living with HIV, HCV- hepatitis c virus, HBV- hepatitis b virus, CA-carcinoma, BA- bronchial asthma, CAD- coronary artery disease, HL-Hodgkin's lymphoma)

8. SUBSTANCE ADDICTION

Substance	No. of cases	Percentage
SMOKE	8	8.0%
ALCOHOL	17	17.0%
OPIOID	1	1.0%
TOBACCO	1	1.0%

Table 8- Distribution of cases as per history of substance addiction

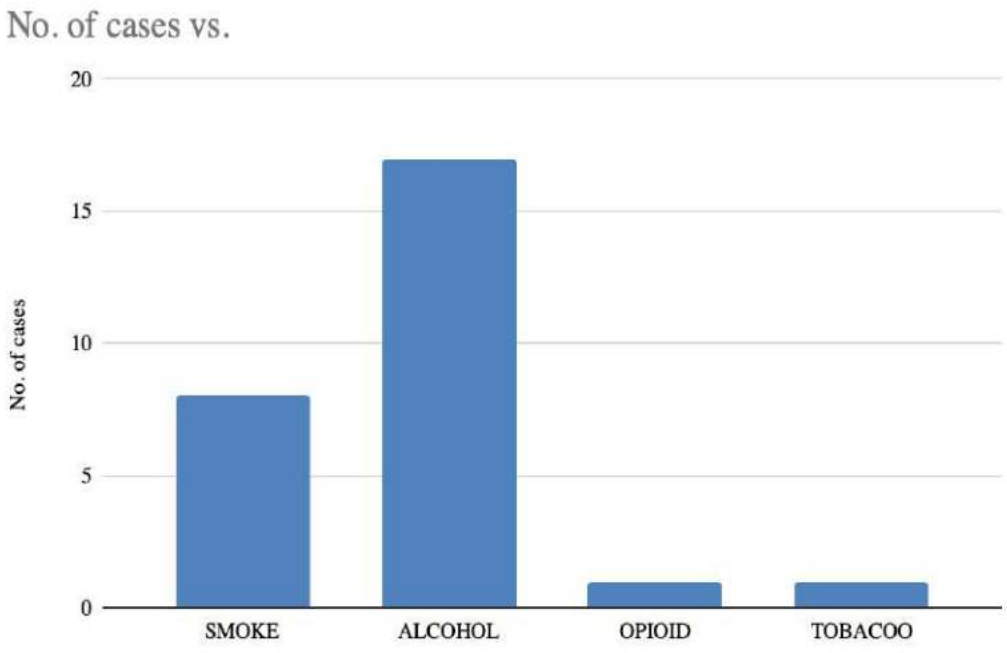

Figure 8-Distribution of cases as per history of substance addiction

9. GENERAL PHYSICAL EXAMINATION

Physical signs/vitals	No. of patients	percentage
HYPOXIA (SPO_2 <94%)	35	35.0%
ICTERUS	3	3.0%
CLUBBING	3	3.0%
LAP	3	3.0%

Table 9- General physical examination at admission (SPO_{2-} - oxygen saturation, LAP- Lymphadenopathy)

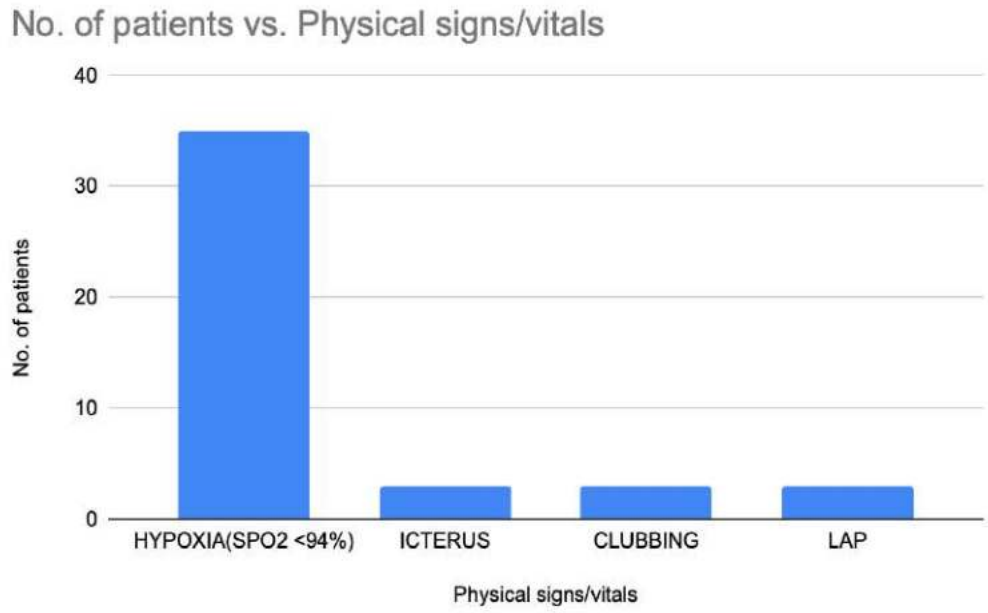

Figure 9 -General physical examination at admission (SPO_{2-} - oxygen saturation, LAP- Lymphadenopathy)

10. CHEST EXAMINATION

SIGN		no.of cases	percentage
SYMMETRY	AS	81	81%
	S	19	19%
TRACHEA	C	30	30%
	SHIFTED	70	70%
	DECREASED	100	100%
	DULL	100	100%
AUSCULTATION	LEFT ABSENT	42	42%
	RIGHT ABSENT	51	51%
	B/L ABSENT		7%
TOTAL		100	100%

Table10- Chest examination of patients with exudative pleural effusion (AS- asymmetrical, S- symmetrical, C-central, B/L- bilateral)

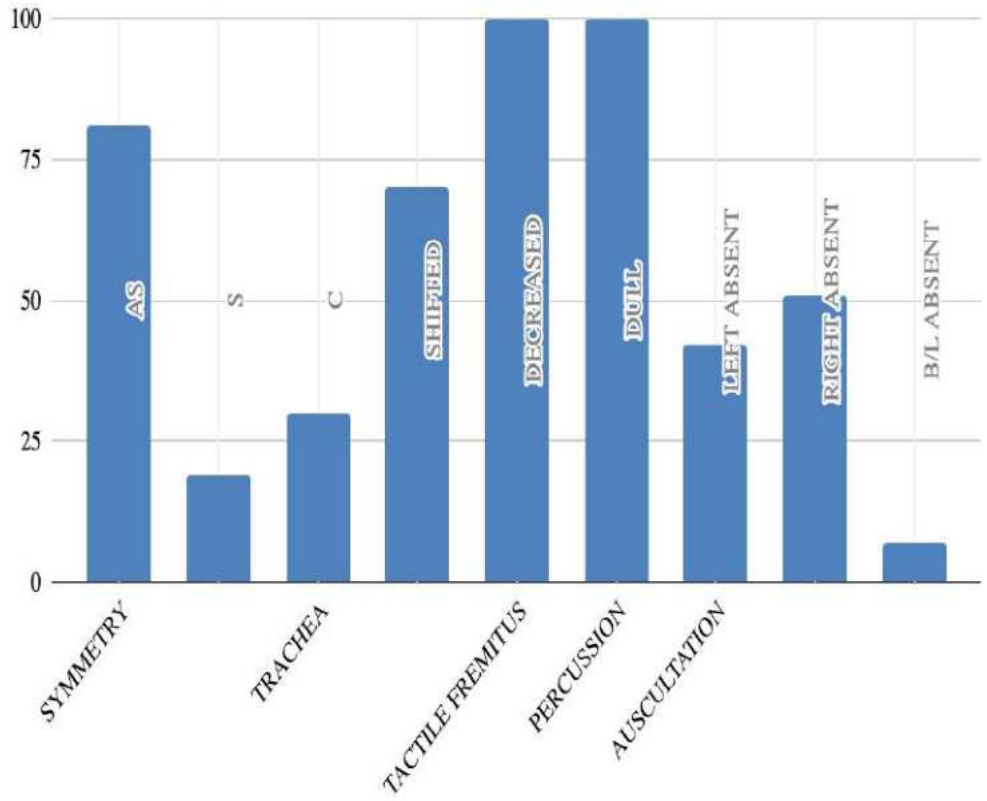

Figure 10- Distribution of physical findings of chest examination. X axis denotes physical findings and y axis denotes percentage (AS- asymmetrical, S- symmetrical, C-central, B/L- bilateral).

11. OTHER SYSTEMIC EXAMINATION

SYSTEM	SIGN	NO.OF CASES	
CVS	MUFFLED HEART SOUNDS	1	1%
CNS	ENCEPHALOPATHY	7	7%
ABDOMEN	ASCITIS	7	7%
	RUQ TENDER	1	1%
OTHER	CHOROID TUBERCLES	1	1%

TABLE 11- Percentage of other systemic findings (CVS-Cardiovascular system, CNS-Central nervous system, RUQ- right upper quadrant)

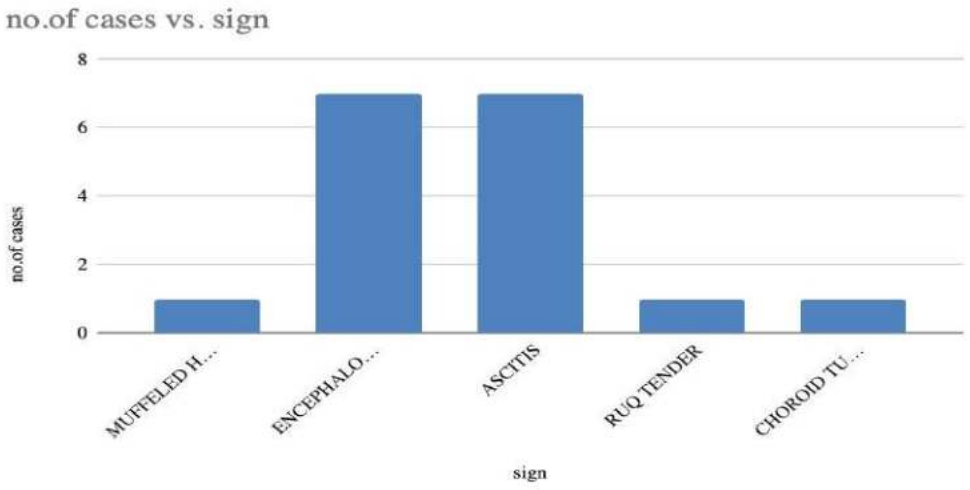

Figure 11- Percentage of other systemic findings (CVS-Cardiovascular system, CNS-Central nervous system, RUQ- right upper quadrant)

RADIOLOGICAL TEST

12. CHEST X RAYS

Laterality	No. of cases	Percentage
B/L	7	7.0%
LT	42	42.0%
RT	51	51.0%
Total	100	100.0%

Table 12- Laterality on chest X ray (B/L- bilateral, LT- left, RT- right)

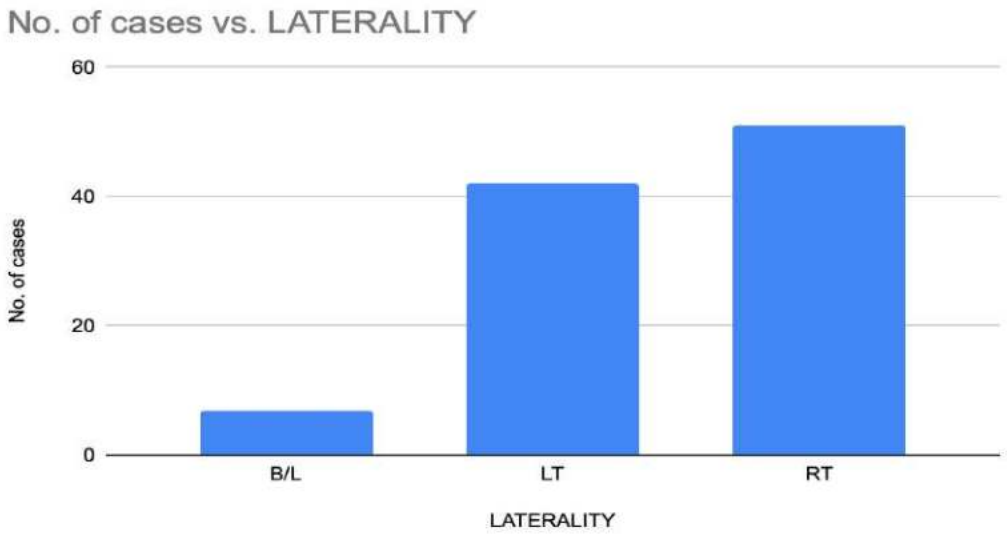

Figure 12- Laterality on chest X ray (B/L- bilateral, LT- left, RT- right)

Table 13- Other findings on chest x ray

Finding	No. of cases	Percentage
OPACITY	21	21%
MEDIASTINAL SHIFT	76	76%
CAVITY	3	3%
FIBROSIS	3	3%
GROUND GLASS OPACITY	1	1%
MILIARY MOTTLING	1	1%
PNEUMOTHORAX	1	1%
RIB FRACTURE	1	1%
Total	100	100%

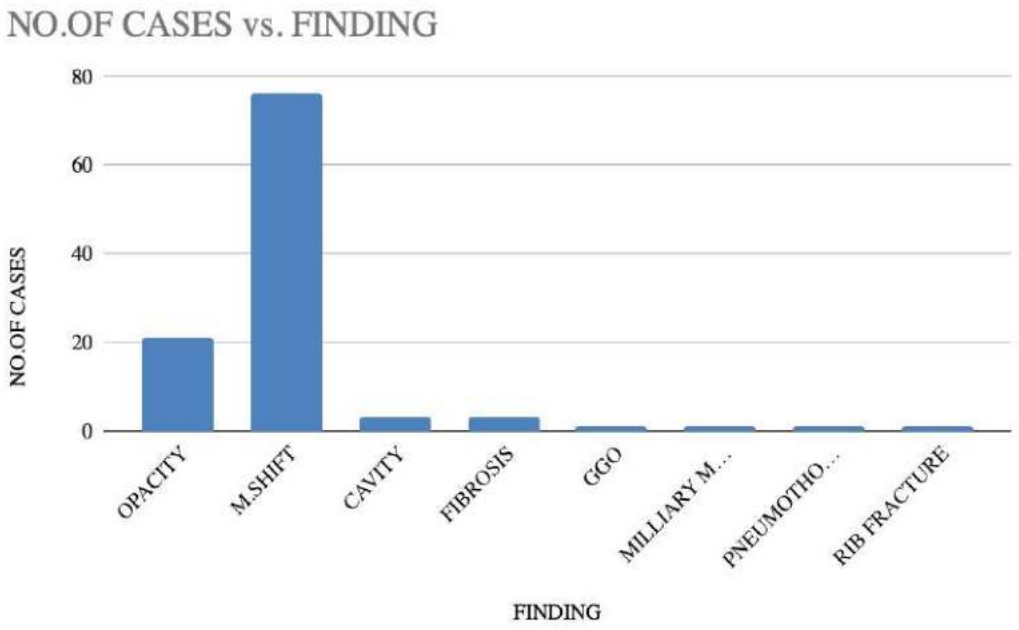

Figure 13 - Other findings on chest X ray (GGO- Ground glass opacities, M.shift- mediastinal shift)

14. ULTRASONOGRAPHY CHEST

USG CHEST_LATERALITY	No. of cases	Percentage
B/L	7	7.0%
LT	42	42.0%
RT	51	51.0%
Total	100	100.0%

Table 14 - Laterality on USG chest (B/L- Bilateral, RT- right, LT- left)

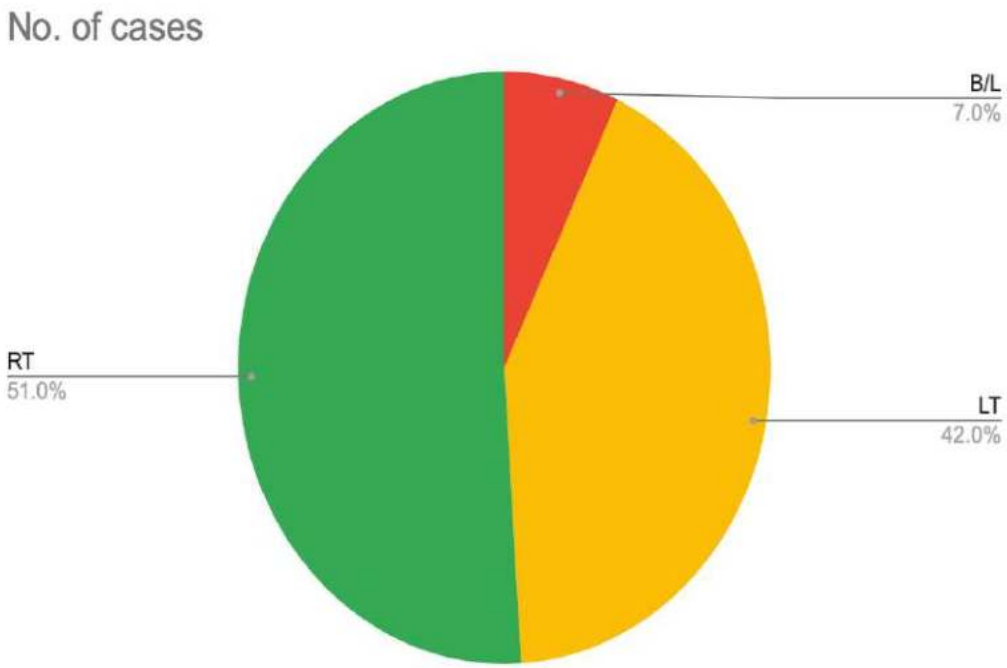

Figure 14- Laterality on USG chest (B/L- Bilateral, RT- right, LT- left)

Table 15- Other findings on USG chest

FINDING	NO.OF CASES	PERCENTAGE
MILD	21	21%
MODERATE	59	59%
SIGNIFICANT	20	20%
SEPTAE/MULTILOCULATED	17	17%
SINGLE LOCULATION	13	13%

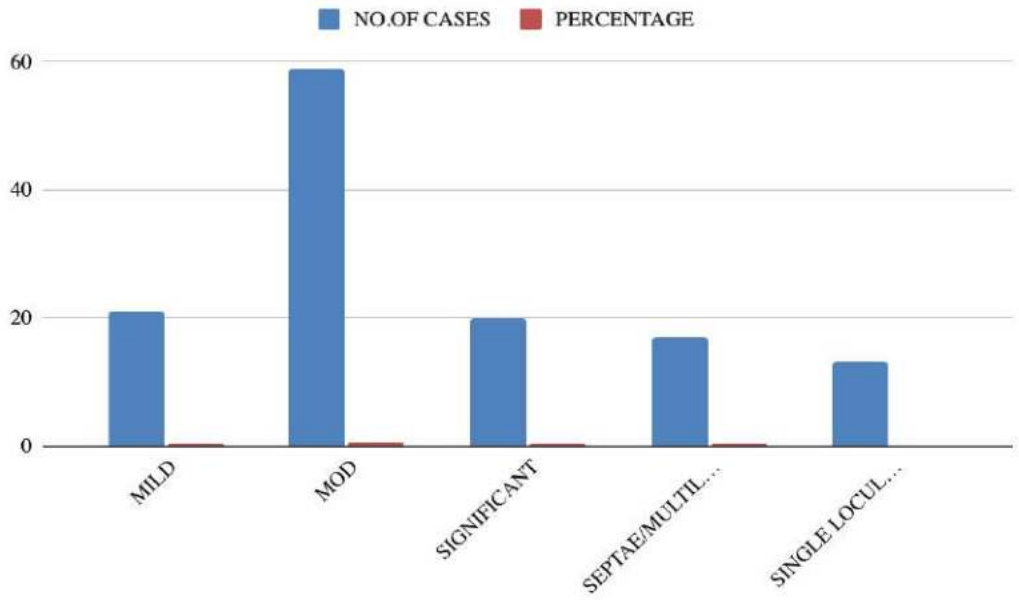

Figure 15- Other findings on USG chest (X axis-finding, Y axis-Number of cases)

16. COMPUTED TOMOGRAPHY CHEST

		No. of cases	Percentage
LATERALITY	RT SIDED	11	40.07%
	LT SIDED	13	48.14%
	B/L	3	11.11%
GRADING	MILD	3	11.11%
	MOD	16	59.25%
	SIGNIFICANT	8	29.62%
OTHERS	CA LUNG	4	14.81%
	GGO	2	7.4%
	P.EMBOLISM	3	11.11%
	CONSOLIDATION	1	3.7%
	HYDROPNEUMOTHORAX	1	3.7%
	EMPYEMA	3	11.11%
	TB	1	3.7%
	CAVITY	1	3.7%
	NEC LAP	1	3.7%
	MASS	2	7.4%
TOTAL		27	

Table 16 - CT chest findings (RT- right, LT- left, B/L- bilateral, MOD- moderate, CA- carcinoma, GGO- ground glass opacities, P.embolism- pulmonary embolism, TB- tuberculosis, NEC LAP- necrotising lymphadenopathy)

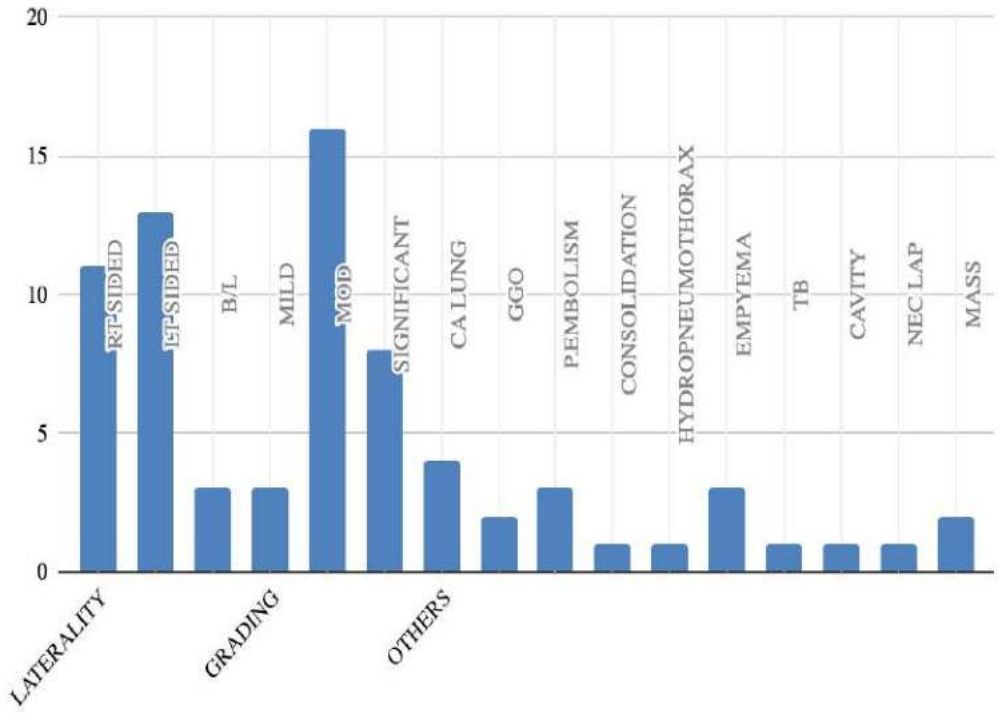

Figure 16- CT chest findings (RT- right, LT- left, B/L- bilateral, MOD- moderate, CA- carcinoma, GGO- ground glass opacities, P.embolism- pulmonary embolism, TB- tuberculosis, NEC LAP- necrotising lymphadenopathy)

17. USG GUIDED THORACOCENTESIS

PROCEDURE	NO. OF CASES	PERCENTAGE
USG GUIDED DX TAP	100	100%
USG GUIDED THX TAP	28	28%
COMPLICATIONS	4	4%
HYDROPNEUMOTHORAX	3	3%
HEMOTHORAX	1	1%
Total	100	100%

Table17- USG Guided thoracentesis (DX- diagnostic, THX- therapeutic, USG-ultrasonography)

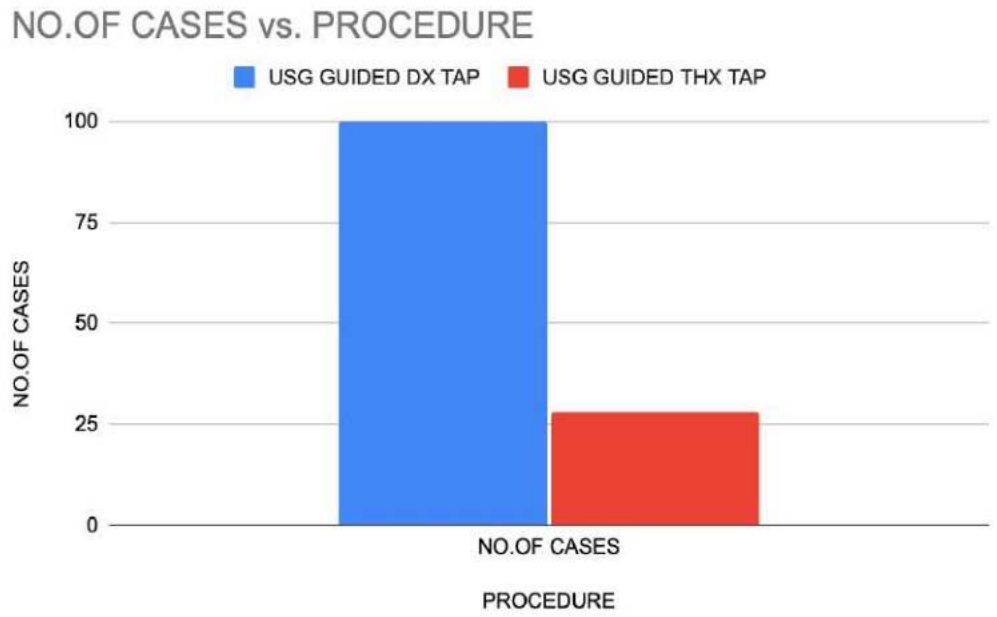

Figure-17 USG Guided thoracentesis (DX- diagnostic, THX- therapeutic, USG-ultrasonography)

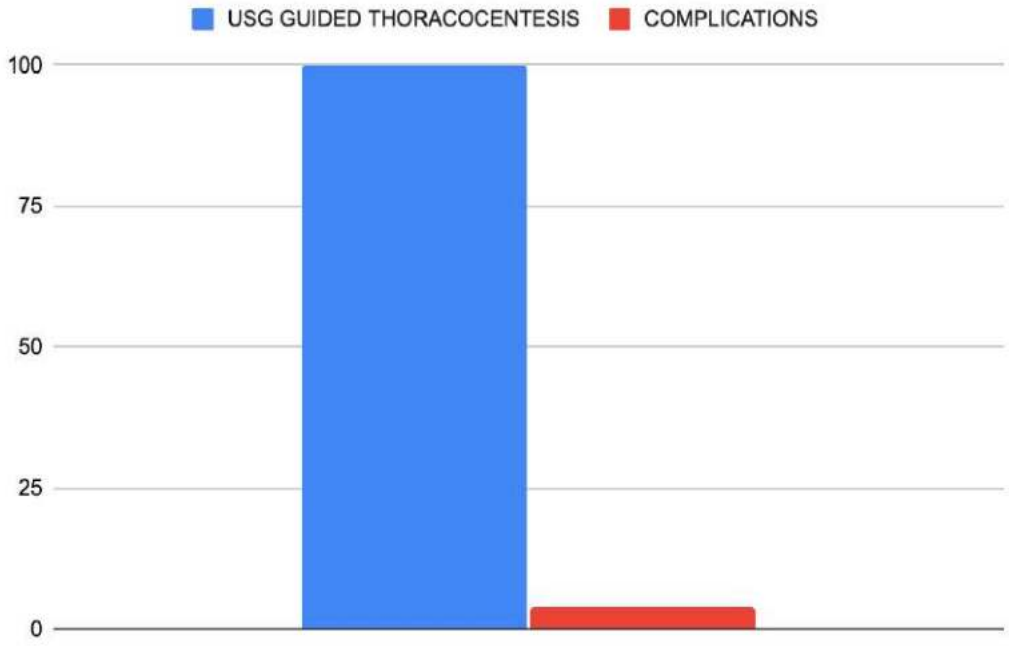

Figure 18 -USG Guided thoracentesis and complications.

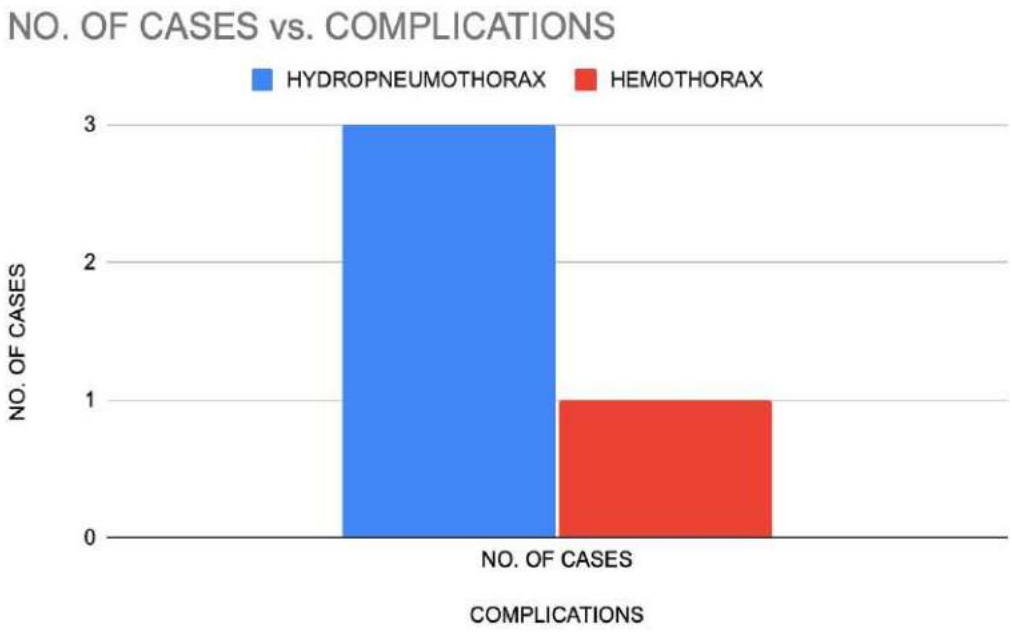

Figure 19-Detailed complications in USG guided thoracentesis

PLEURAL FLUID ANALYSIS

18. APPEARANCE/ COLOR OF PLEURAL FLUID

Color	No. of cases	Percentage
HEMORRHAGIC	11	11%
STRAW	64	64%
PUS	22	22%
CHYLOUS	1	1%
ANCHOVY	2	2%
Total	100	100%

Table 18 - Pleural fluid appearance/color

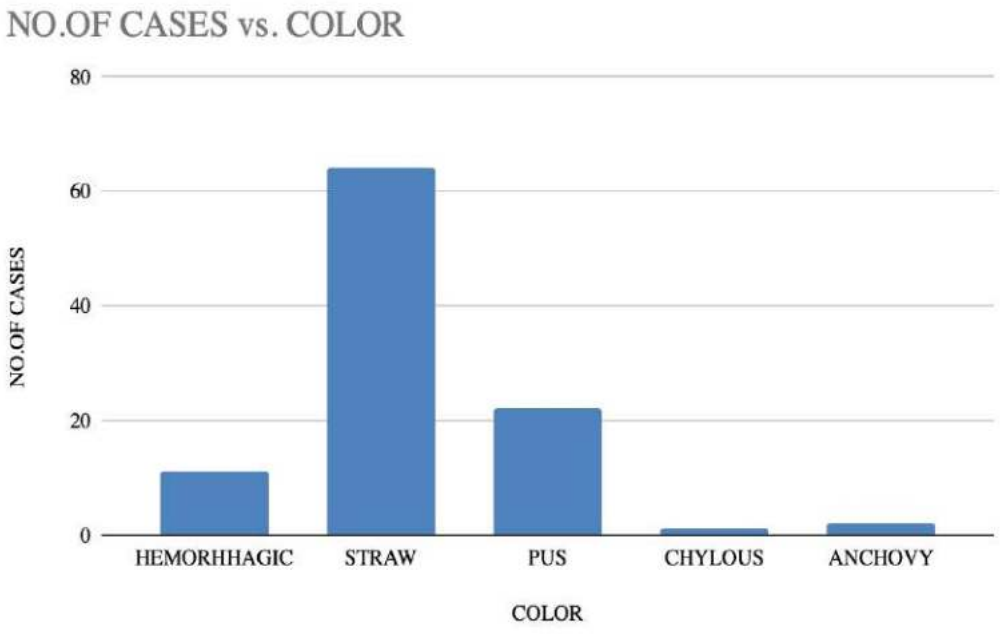

Figure 20- Pleural fluid appearance/color

19. ETIOLOGICAL CLASSIFICATION

Etiology	No. of cases	Percentage
TB	42	42.0%
Malignant	9	9.0%
Empyema	15	15.0%
Parapneumonic	16	16.0%
Others	11	11.0%
Unknown	7	7.0%
Total	100	100.0%

Table 19- Etiology of exudative pleural effusion (TB- tuberculosis).

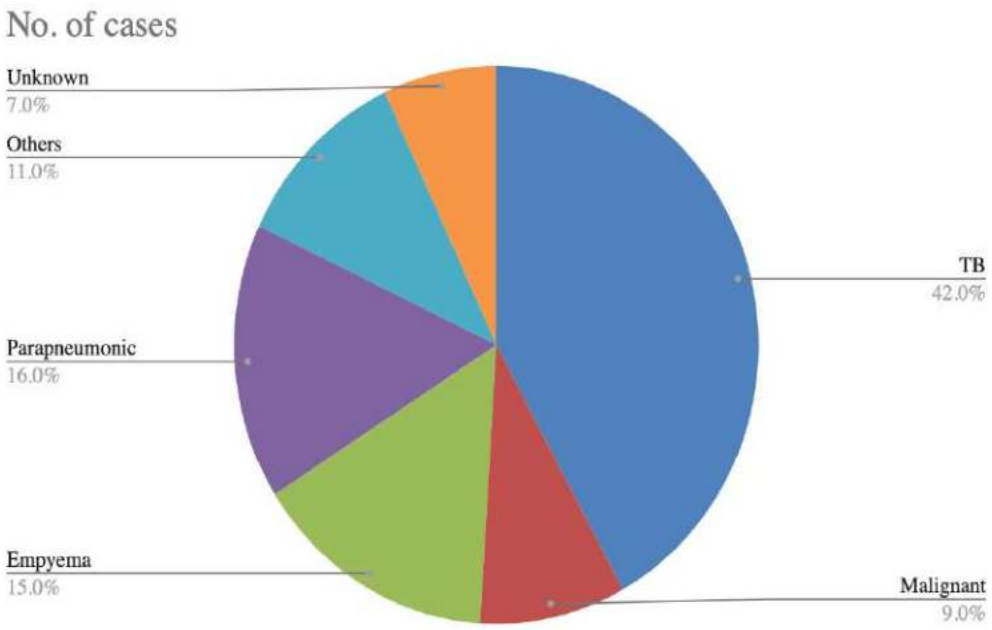

Figure 21- Etiological class of exudative pleural effusion (TB- tuberculosis).

Etiology	No. of cases	Percentage
Tb P.E Probable	28	28.0%
Tb P.E Confirmed	8	8.0%
Tb Empyema Definitive	4	4.0%
Tb Empyema Probable	2	2.0%
M. P.E Probable	2	2.0%
M. P.E Confirmed	7	7.0%
Empyema Simple	6	6.0%
Empyema Complex	9	9.0%
Pp.E Typical	1	1.0%
Pp.E Borderline	0	0.0%
Pp.E Simple.Complicated	7	7.0%
Pp.E Complex.Complicated	8	8.0%
Chylothorax	1	1.0%
Hemothorax	3	3.0%
Pulmonary Embolism	3	3.0%
Viral	2	2.0%
Parasitic	2	2.0%
Unknown	7	7.0%

Table 20- Detailed etiological classification (TB P.E- tubercular pleural effusion, M.PE- Malignant pleural effusion,PP.E- parapneumonic pleural effusion)

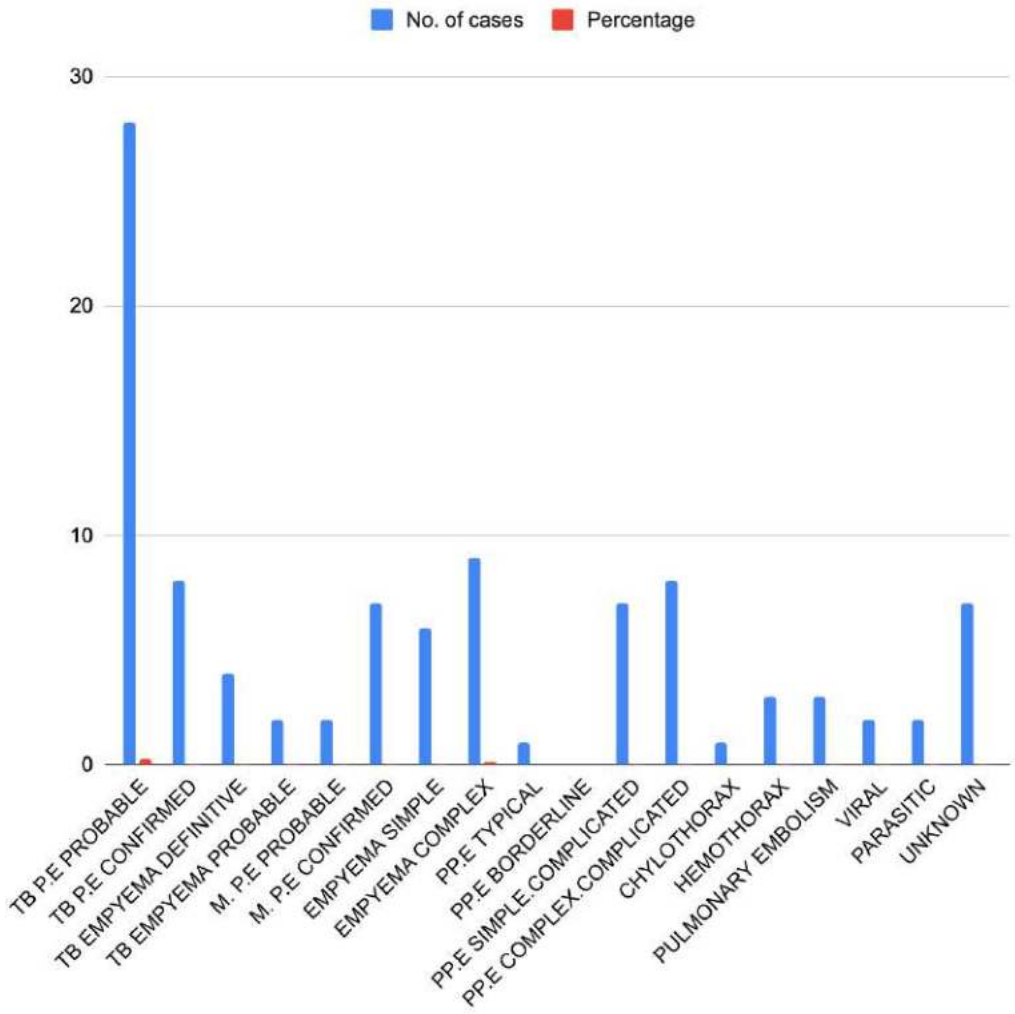

Figure 22- Detailed etiological classification (TB P.E- tubercular pleural effusion, M.PE- Malignant pleural effusion, PP.E- parapneumonic pleural effusion)

Table 21- Distribution as per the gross appearance and etiology (TB-Tuberculosis)

Etiology	TB	Malignant	Empyema	Parapneumonic	Others	Unknown	Total
HEMORRHAGIC	0	3	0	0	5	3	11
STRAW	35	6	0	16	3	4	64
PUS	7	0	15	0	0	0	22
CHYLOUS	0	0	0	0	1	0	1
ANCHOVY.SAUCE	0	0	0	0	2	0	2
Total	42	9	15	16	11	7	100

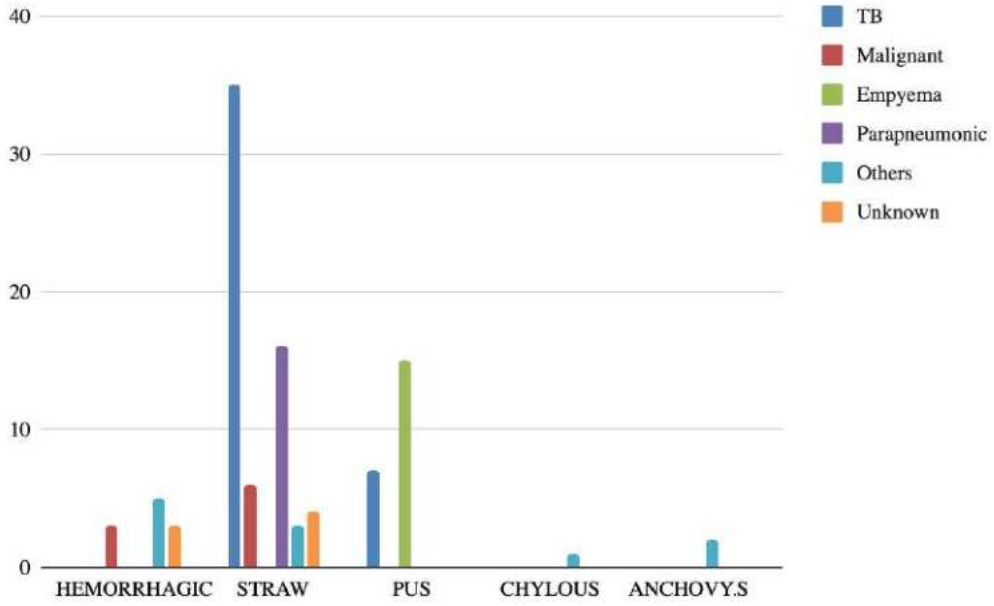

Figure 23 - Distribution as per the gross appearance and etiology (TB-Tuberculosis)

etiology	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum	F	p-value
					Lower Bound	Upper Bound				
TB	42	519.05	434.342	67.020	383.70	654.40	100	2200	2.923	0.017
Malignant	9	588.89	742.789	247.596	17.93	1159.85	100	2500		
Empyema	15	1911.33	2570.444	663.686	487.87	3334.80	100	10000		
Parapneumonic	16	1238.75	1073.057	268.264	666.96	1810.54	90	3800		
Others	11	955.45	2216.490	668.297	-533.60	2444.51	10	7600		
Unknown	7	300.00	282.843	106.904	38.41	561.59	100	800		
Total	100	882.00	1414.910	141.491	601.25	1162.75	10	10000		

Table 22- Distribution of mean TLC according to etiology (TB- tuberculosis, Total leukocyte count, N- total no of cases)

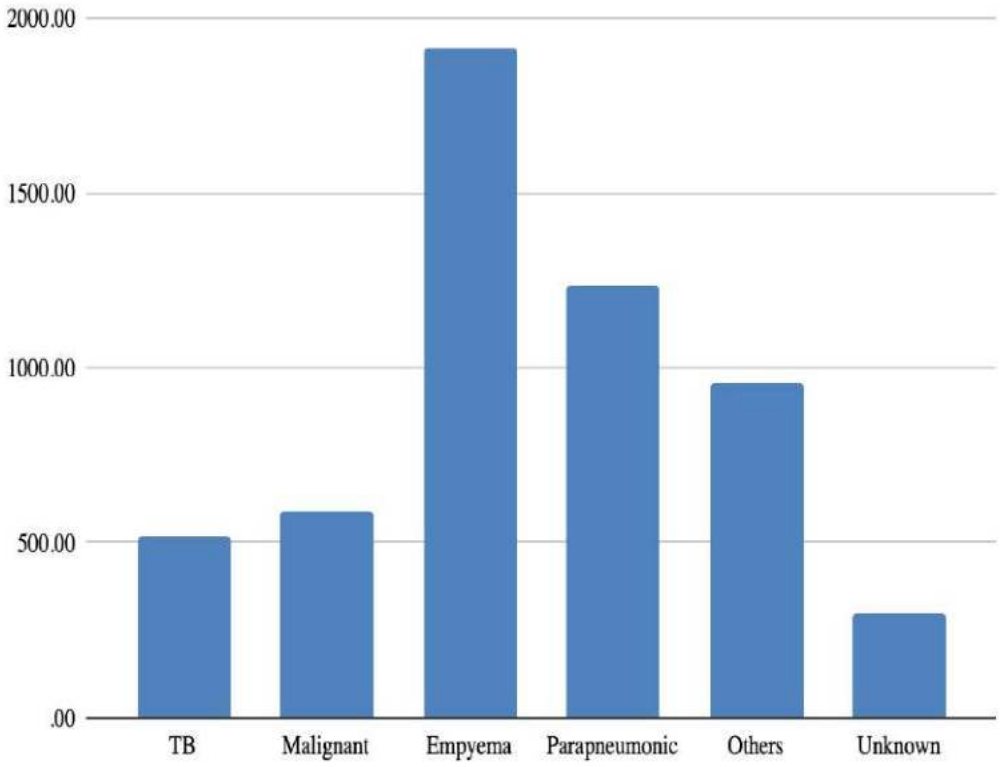

Figure 24- Distribution of mean TLC according to etiology (TLC- Total leukocyte count, TB- tuberculosis,)

		TLC cells/mm^3			Total	Chi-square value	p-value
		< 2500	2500-5000	5000-10000			
Etiology	TB	42	0	0	42	16.338	0.09
	Malignant	8	1	0	9		
	Empyema	12	2	1	15		
	Parapneumonic	13	3	0	16		
	Others	10	0	1	11		
	Unknown	7	0	0	7		
Total		92	6	2	100		

Table 23 - Association of TLC according to etiology (TLC- Total leukocyte count, TB- tuberculosis)

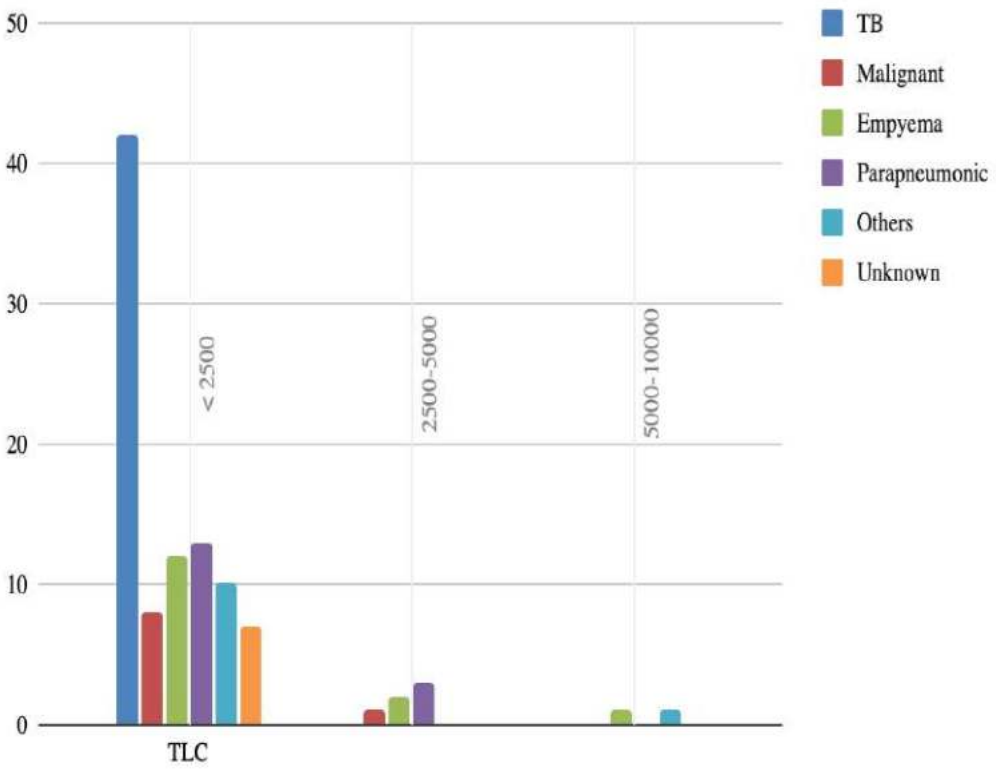

Figure 25- Association of TLC with etiology (TLC- Total leukocyte count, TB- tuberculosis)

		L		NE		Total
		No. of cases	%age	No. of cases	%age	
Etiology	TB	40	95.24%	2	4.76%	42
	Malignant	8	88.89%	1	11.11%	9
	Empyema	2	13.33%	13	86.67%	15
	Parapneumonic	2	12.50%	14	87.50%	16
	Others	6	54.55%	5	45.45%	11
	Unknown	3	42.86%	4	57.14%	7
Total		61	61.00%	39	39.00%	100

Table 24- Distribution as per pleural fluid differential cell count and etiology (L- Lymphocyte, NE- neutrophil)

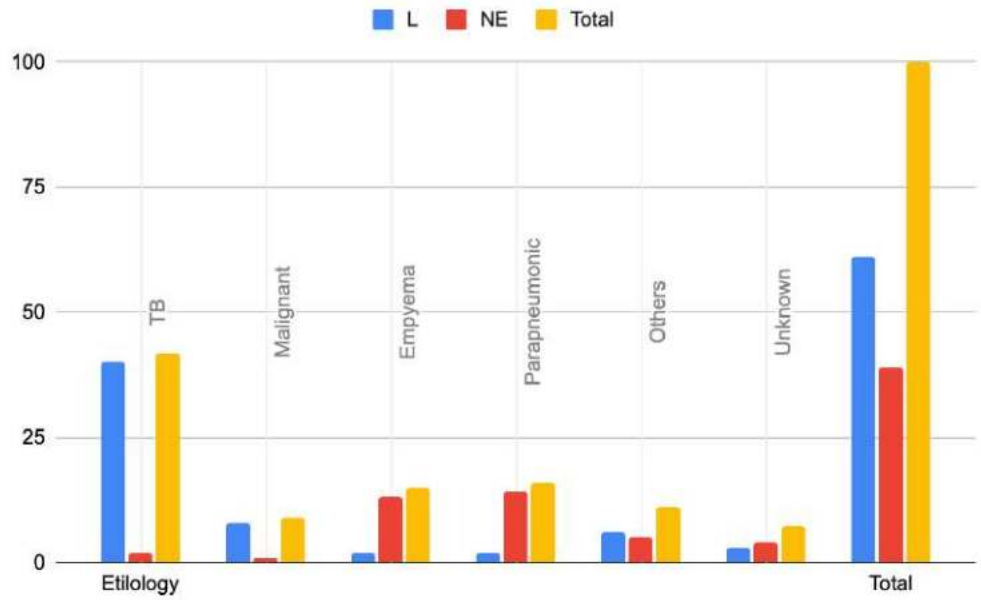

Figure 26- Distribution as per pleural fluid differential cell count and etiology (L- Lymphocyte, NE- neutrophil)

		L/N RATIO		Total	Chi-square value	p-value
		< 0.75	> 0.75			
Etiology	TB	4	38	42	46.438	0.0001
	Malignant	2	7	9		
	Empyema	13	2	15		
	Parapneumonic	13	3	16		
	Others	5	6	11		
	Unknown	1	6	7		
Total		38	62	100		

Table 25 - Association of pleural fluid lymphocyte to neutrophil ratio (L/N- lymphocyte to neutrophil ratio, TB- tuberculosis) and etiology

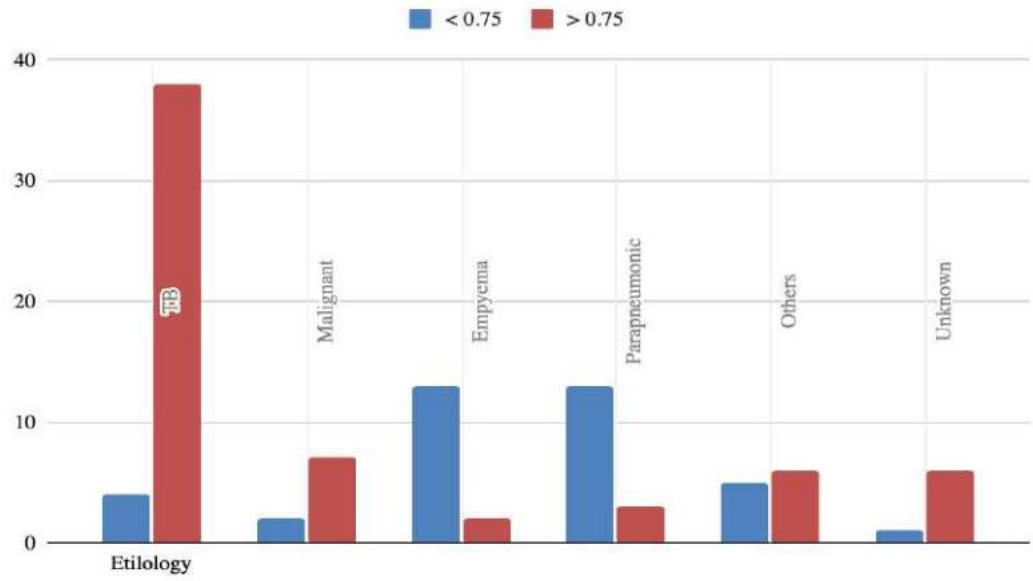

Figure 27 - Association of pleural fluid lymphocyte to neutrophil ratio (L/N- lymphocyte to neutrophil ratio, TB- tuberculosis) and etiology

Table 26- Sensitivity and Specificity of Pleural Fluid Lymphocyte to Neutrophil Ratio > 0.75 to Diagnose Tuberculous Pleural Effusion (TB-tuberculosis)

		L/N RATIO		**Total**
		< 0.75	**> 0.75**	
TB	Y	4	38	42
	N	34	24	58
Total		38	62	100
Statistic	Value	95% CI		
Sensitivity	61.29%	48.07% to 73.40%		
Specificity	89.47%	75.20% to 97.06%		
Positive Likelihood Ratio	5.82	2.26 to 15.02		
Negative Likelihood Ratio	0.43	0.31 to 0.60		
Disease prevalence (*)	62.00%	51.75% to 71.52%		
Positive Predictive Value (*)	90.48%	78.64% to 96.08%		
Negative Predictive Value (*)	58.62%	50.42% to 66.37%		
Accuracy (*)	72.00%	62.13% to 80.52%		

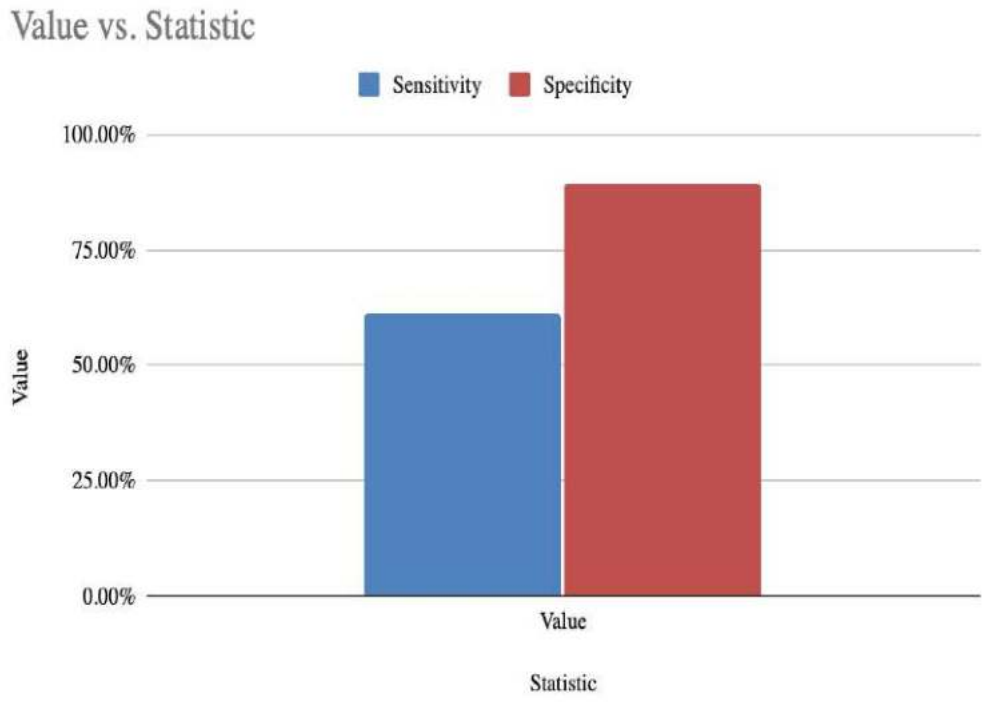

Figure 28 - Sensitivity and Specificity of Pleural Fluid Lymphocyte to Neutrophil Ratio > 0.75 to Diagnose Tuberculous Pleural Effusion

Table 27 - Distribution of subjects according to pleural fluid ADA and etiology (TB-tuberculosis, ADA- adenosine deaminase)

		ADA(U/L)			Total	Chi-square value	p-value
		< 40	40-60	> 60			
Etiology	TB	0	2	40	42	62.566	0.0001
	Malignant	4	4	1	9		
	Empyema	7	2	6	15		
	Parapneumonic	4	6	6	16		
	Others	5	4	2	11		
	Unknown	6	1	0	7		
Total		26	19	55	100		

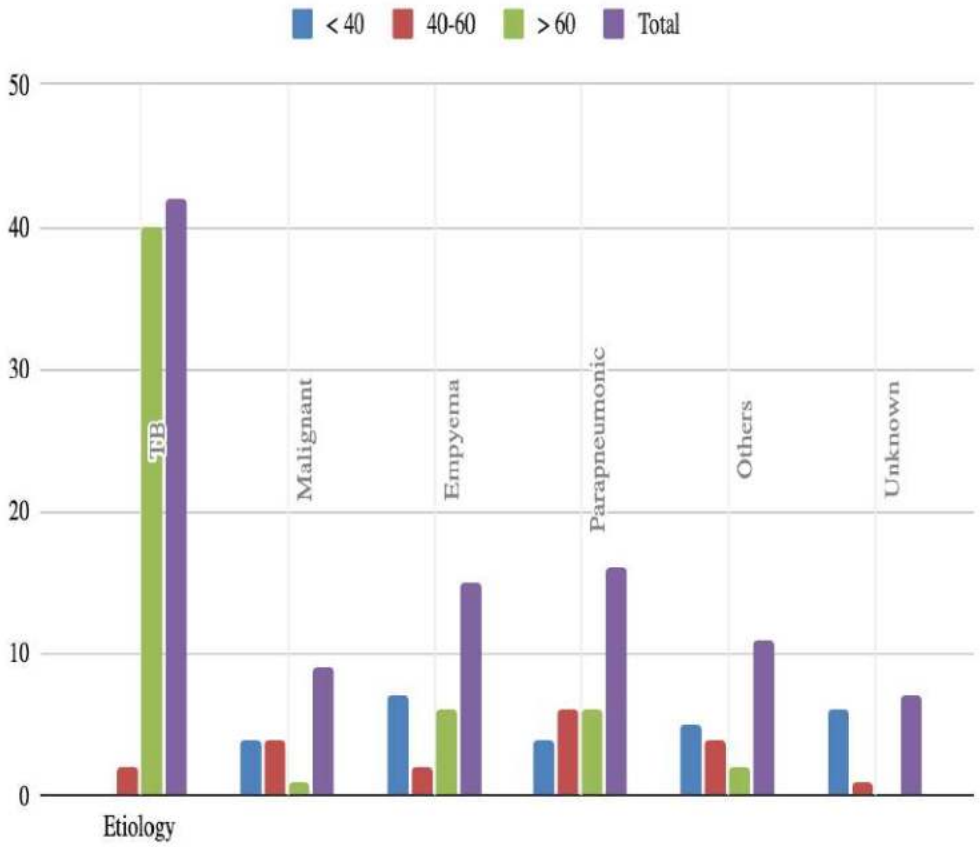

Figure 29 - Distribution of subjects according to pleural fluid ADA and etiology (TB-tuberculosis, ADA- adenosine deaminase)

Table 28 - Association of Pleural Fluid Adenosine Deaminase and Tuberculous and Malignant Pleural Effusion (TB-tuberculosis, ADA-adenosine deaminase)

		ADA (U/L)		Total	Chi-square value	p-value
		< 40	> 40			
TB	Y	0	42	42	25.443	0.0001
	N	26	32	58		
Malignant	Y	4	5	9	1.749	0.186
	N	22	69	91		
Total		26	74	100		

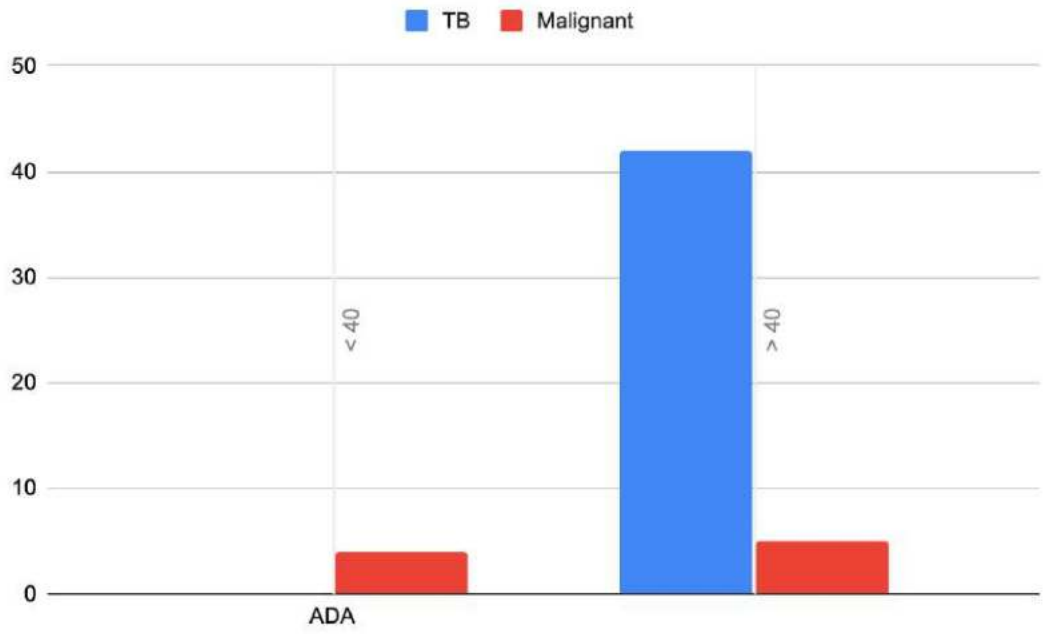

Figure 30 - Association of Pleural Fluid Adenosine Deaminase and Tuberculous and Malignant Pleural Effusion (TB-tuberculosis, ADA-adenosine deaminase)

Statistic	**Value**	**95% CI**
Sensitivity	56.76%	44.72% to 68.23%
Specificity	100.00%	86.77% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	0.43	0.33 to 0.56
Disease prevalence (*)	74.00%	64.27% to 82.26%
Positive Predictive Value (*)	100.00%	
Negative Predictive Value (*)	44.83%	38.49% to 51.33%
Accuracy (*)	68.00%	57.92% to 76.98%

Table 29 -Sensitivity and Specificity of ADA to Diagnose Tubercular Pleural Effusion

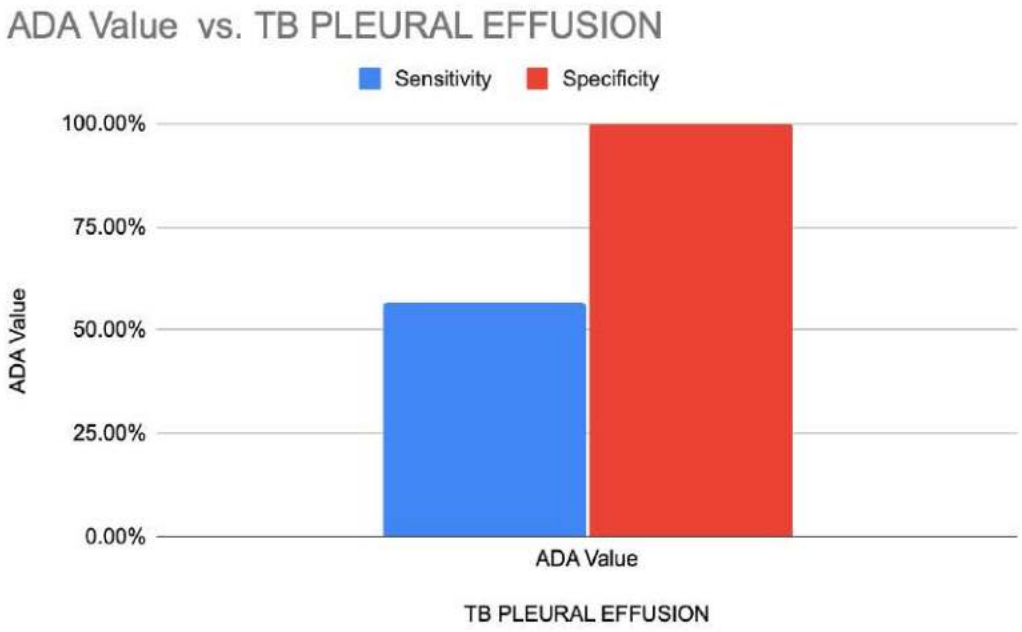

Figure 31- Sensitivity and Specificity of ADA to Diagnose Tubercular Pleural Effusion

Table 30 - Distribution according to combined ADA and L/N ratio and etiology (ADA-adenosine deaminase, L/N- lymphocyte to neutrophil ratio)

ADA >40 (U/L)& L/N ratio >0.75	No. of cases	Percentage (%)
TB	38	75.5
Malignant	5	10.2
Empyema	2	4.1
Parapneumonic	1	2.0
Others	4	6.1
Unknown	1	2.0

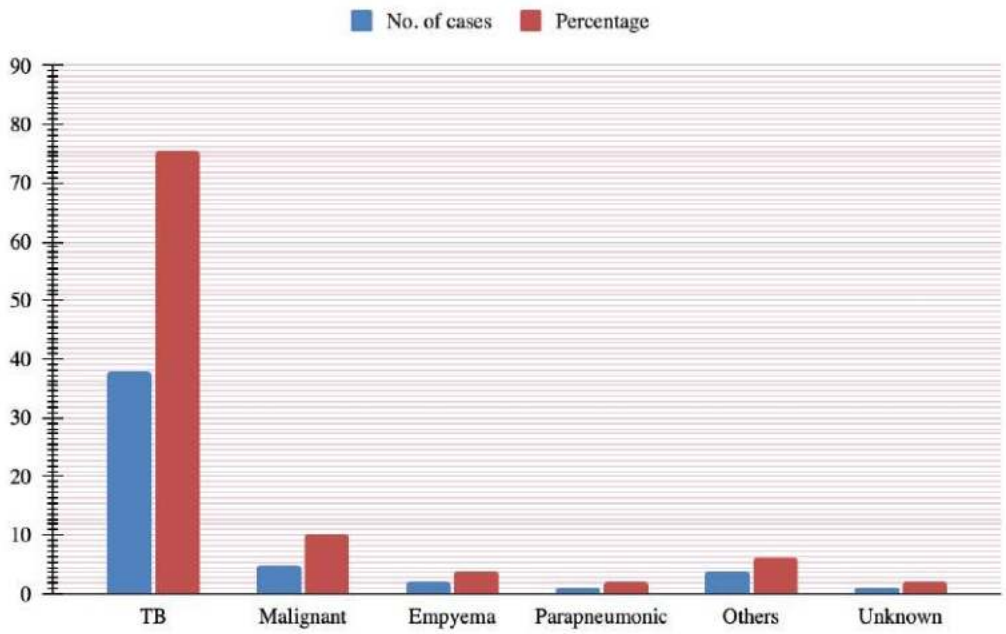

Figure 32- Distribution according to combined ADA and L/N ratio and etiology (ADA-adenosine deaminase, L/N- lymphocyte to neutrophil ratio)

Table 31- Association of combined ADA and L/N ratio and tuberculous pleural effusion (ADA-adenosine deaminase, L/N-lymphocyte to neutrophil ratio, TB- tuberculosis)

		ADA >40(U/L) & L/N ratio >0.75		**Total**
		<No	**Yes**	
TB	Y	4	38	42
	N	45	13	58
Total		49	51	100

Table 32- sensitivity and specificity of combined ADA and L/N ratio in tuberculous pleural effusion (ADA-adenosine deaminase, L/N-lymphocyte to neutrophil ratio, TB- tuberculosis).

Statistic	**Value**	**95% CI**
Sensitivity	74.51%	60.37% to 85.67%
Specificity	91.84%	80.40% to 97.73%
Positive Likelihood Ratio	9.13	3.52 to 23.67
Negative Likelihood Ratio	0.28	0.17 to 0.45
Disease prevalence (*)	51.00%	40.80% to 61.14%
Positive Predictive Value (*)	90.48%	78.56% to 96.10%
Negative Predictive Value (*)	77.59%	68.25% to 84.79%
Accuracy (*)	83.00%	74.18% to 89.77%

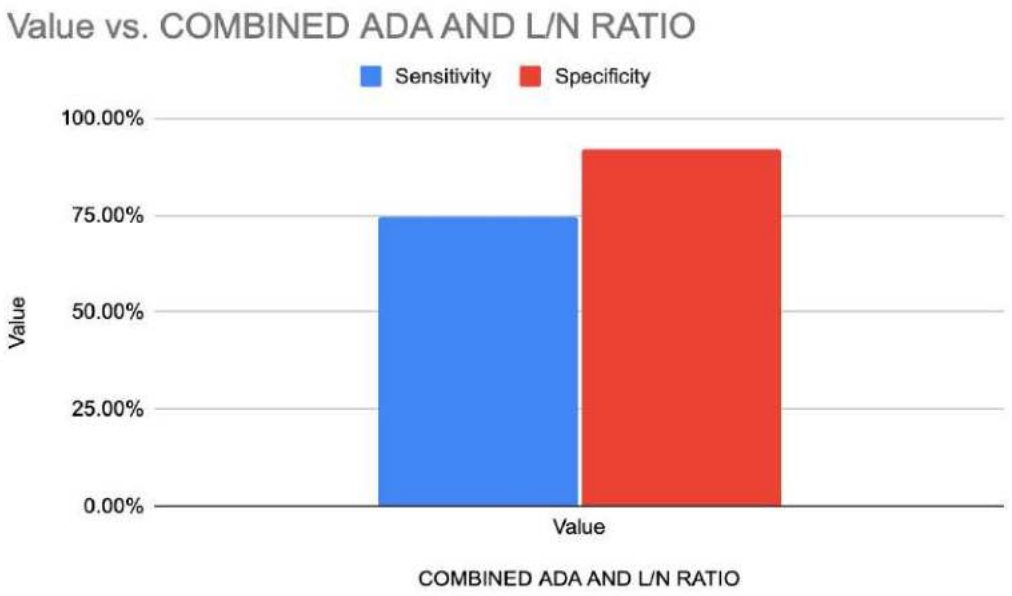

Figure 33 -Association of combined ADA and L/N ratio and tuberculous pleural effusion (ADA-adenosine deaminase, L/N-lymphocyte to neutrophil ratio, TB- tuberculosis).

Etiology	N	Mean Glucose (mg/dl)	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum	F	p-value
					Lower Bound	Upper Bound				
TB	42	87.31	50.904	7.855	71.45	103.17	2	293	8.728	0.0001
Malignant	9	110.22	39.038	13.013	80.22	140.23	72	176		
Empyema	15	36.87	45.066	11.636	11.91	61.82	2	176		
Parapneumonic	16	28.13	22.853	5.713	15.95	40.30	1	90		
Others	11	81.09	23.918	7.212	65.02	97.16	29	114		
Unknown	7	87.57	12.367	4.674	76.13	99.01	65	101		
Total	100	71.67	48.850	4.885	61.98	81.36	1	293		

Table 33 - Distribution of Subjects According to Etiology and mean Pleural Fluid Glucose Levels (N- no of cases, TB - tuberculosis)

		Glucose (mg/dl)			Total	Chi-square value	p-value
		< 40	**40-100**	**>100**			
Etiology	TB	4	30	8	42	46.723	0.0001
	Malignant	0	6	3	9		
	Empyema	10	4	1	15		
	Parapneumonic	12	4	0	16		
	Others	1	8	2	11		
	Unknown	0	6	1	7		
Total		27	58	15	100		

Table 34- Association between Pleural Fluid Sugar Level and Etiology (TB- tuberculosis)

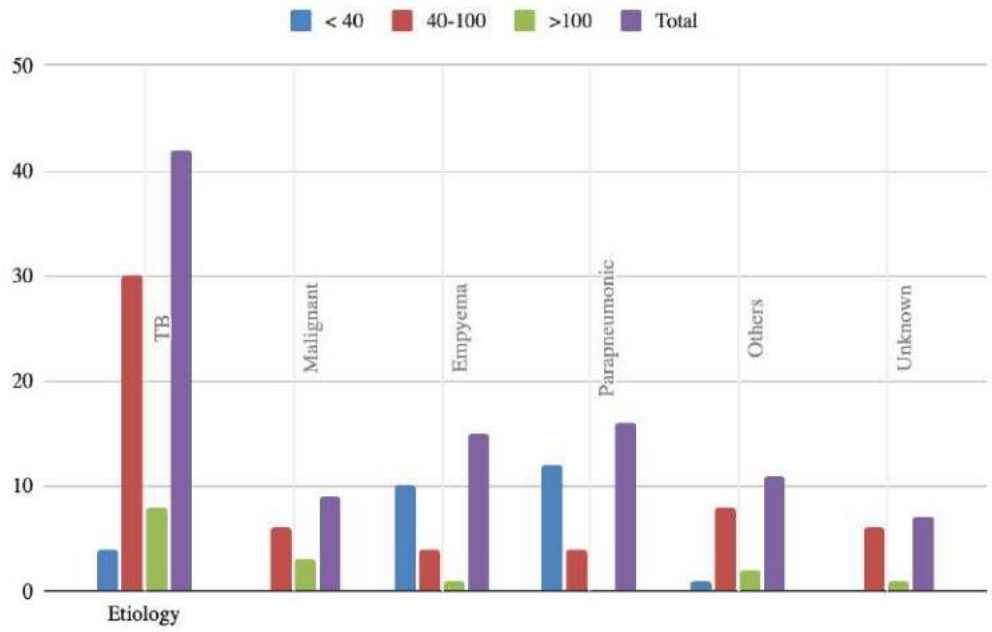

Figure 34- Association between Pleural Fluid Sugar Level and Etiology (TB- tuberculosis)

Table 35- Association between pleural fluid protein to serum protein ratio and etiology (TB- tuberculosis)

		PF: PROTEIN RATIO				Total	Chi-square value	p-value
		< 0.5	0.5-0.7	0.7-0.9	> 0.9			
Etiology	TB	0	24	16	2	42	17.095	0.313
	Malignant	0	5	4	0	9		
	Empyema	0	7	6	2	15		
	Parapneumonic	2	8	6	0	16		
	Others	0	8	2	1	11		
	Unknown	0	5	2	0	7		
Total		2	57	36	5	100		

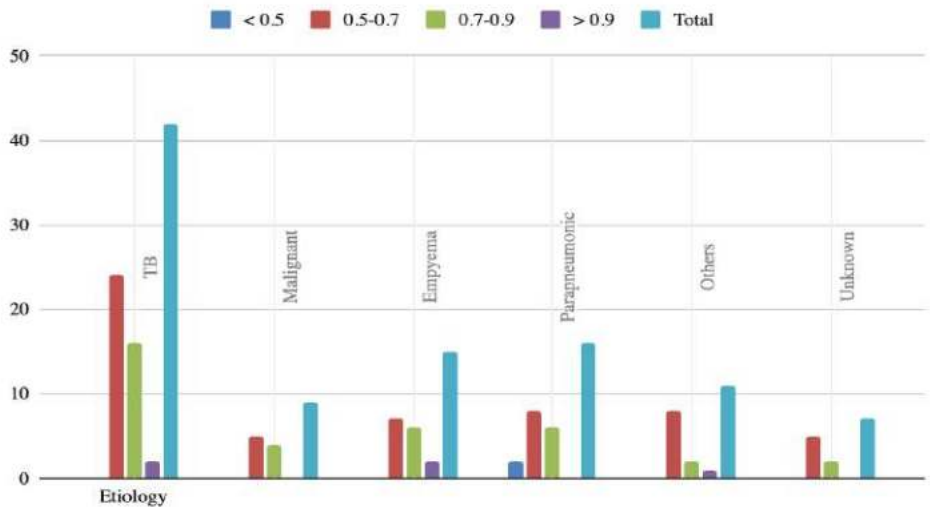

Figure 35- Association between pleural fluid protein to serum protein ratio and etiology (TB- tuberculosis).

Table 36- Association of pleural fluid LDH to serum LDH and etiology. (TB- tuberculosis, LDH- lactate dehydrogenase)

		PF: SLDH RATIO				Total	Chi-square value	p-value
		< 0.6	0.6-1	1-1.5	>1.5			
Etiology	TB	0	8	18	16	42	10.778	0.375
	Malignant	0	2	2	5	9		
	Empyema	0	1	6	7	14		
	Parapneumonic	0	0	8	8	16		
	Others	0	1	7	3	11		
	Unknown	0	0	2	5	7		
Total		0	12	43	44	99		

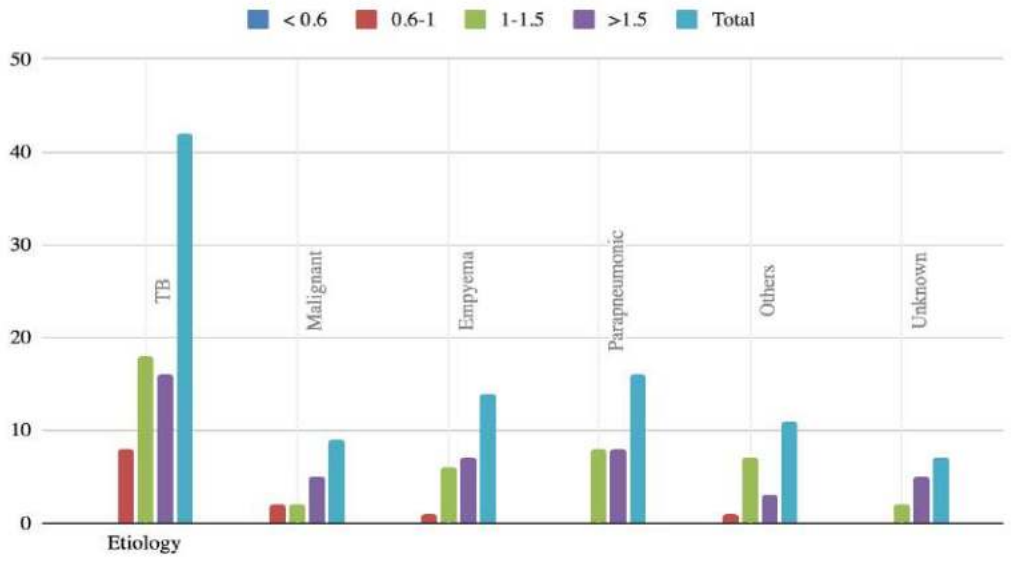

Figure 36- Association of pleural fluid LDH to serum LDH and etiology (LDH- lactate dehydrogenase)

Table 37- Mean serum protein and serum LDH according to etiology

		N	Mean LDH (U/L)	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum	F	p-value
						Lower Bound	Upper Bound				
PROTEIN	TB	42	6.29	.964	.149	5.99	6.59	4	8	0.953	0.451
	malignant	9	6.32	.602	.201	5.86	6.78	6	7		
	empyema	15	6.23	1.022	.264	5.66	6.79	5	8		
	parapneumonic	16	6.22	1.063	.266	5.66	6.79	5	8		
	others	11	5.62	.976	.294	4.96	6.27	4	7		
	unknown	7	6.46	1.282	.484	5.27	7.64	4	8		
	Total	100	6.21	.988	.099	6.01	6.41	4	8		
LDH	TB	42	222.05	123.858	19.112	183.45	260.64	102	712	1.957	0.092
	malignant	9	287.00	134.731	44.910	183.44	390.56	145	540		
	empyema	14	263.57	87.954	23.507	212.79	314.35	150	450		
	parapneumonic	16	214.31	114.397	28.599	153.35	275.27	111	521		
	others	11	299.00	165.268	49.830	187.97	410.03	163	552		
	unknown	7	152.43	36.792	13.906	118.40	186.46	110	200		
	Total	99	236.20	123.496	12.412	211.57	260.83	102	712		

		TB		Total	Chi-square value	p-value
		Y	N			
CBNAAT	NEG	31	58	89	17.068	0.001
	POS	11	0	11		
Total		42	58	100		

Table 38 - Association of CBNAAT and tuberculosis pleural effusion (CBNAAT cartridge based nucleic acid amplification test)

TABLE 39- Sensitivity and specificity of CBNAAT for diagnosing tuberculous pleural effusion (CBNAAT-cartridge based nucleic acid amplification test)

Statistic	**Value**	**95% CI**
Sensitivity	26.19%	13.86% to 42.04%
Specificity	100.00%	93.84% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	0.74	0.62 to 0.88
Disease prevalence (*)	42.00%	32.20% to 52.29%
Positive Predictive Value (*)	100.00%	
Negative Predictive Value (*)	65.17%	60.98% to 69.14%
Accuracy (*)	69.00%	58.97% to 77.87%

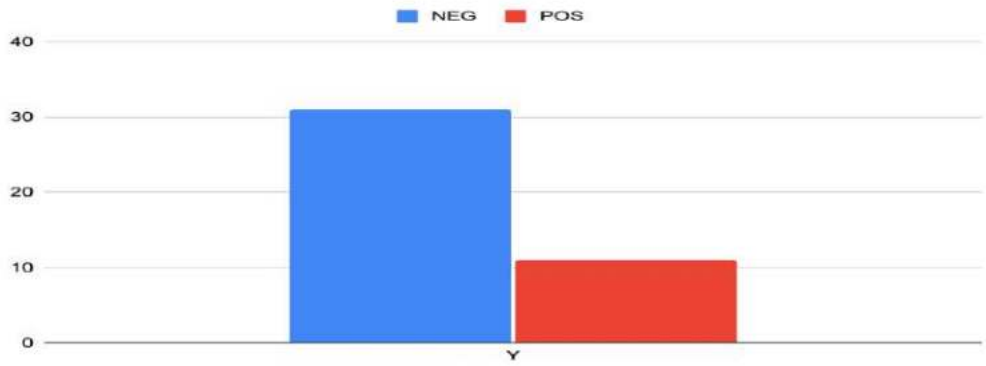

Figure 37- Association of CBNAAT and tuberculous pleural effusion (CBNAAT cartridge based nucleic acid amplification test)

GRAM STAIN	No. of cases	Percentage
GNB	1	1.0%
GNC	2	2.0%
GPC	2	2.0%
NEG	95	95.0%
Total	100	100.0%

Table 40 - percentage of cases positive on Gram's stain (GNB- Gram negative bacilli, GNC- gram negative cocci, GPC- gram positive cocci, NEG- negative)

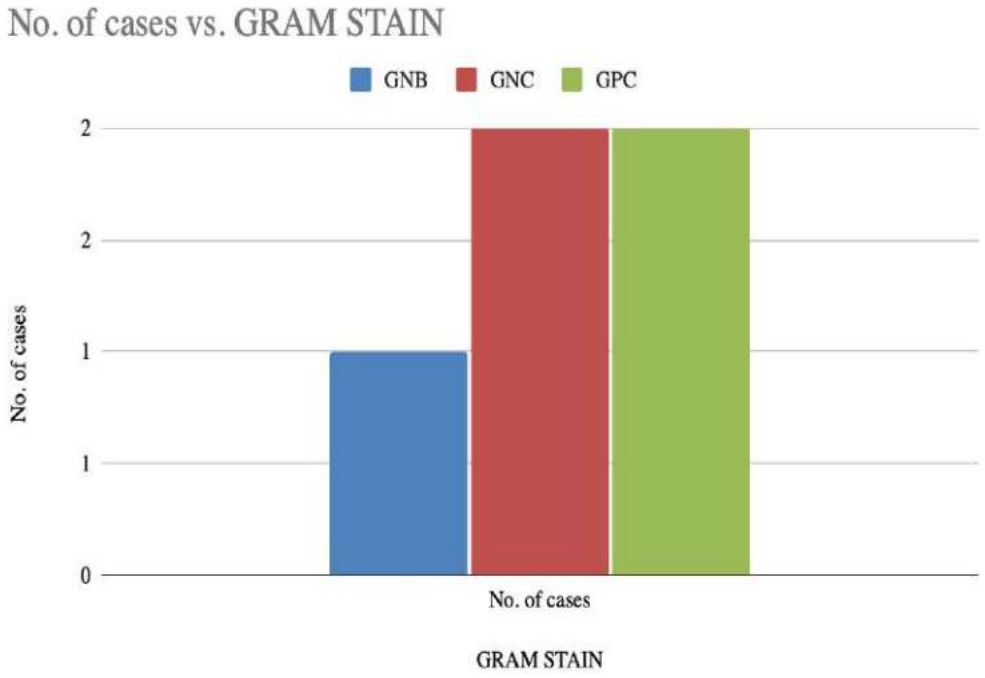

Figure 38 - percentage of cases positive on Gram's stain (GNB- Gram negative bacilli, GNC- gram negative cocci, GPC- gram positive cocci, NEG- negative)

Culture sensitivity	No. of cases	Percentage
ACINETOBACTER	2	2.0%
BURKHOLDERIA	1	1.0%
E.COLI	4	4.0%
K.PNEUM	3	3.0%
MRSA	8	8.0%
P.AERU	2	2.0%
S.PNEUM/ P.AERU	1	1.0%
Total	100	100.0%

Table 41- Percentage of cases with positive culture (e.coli- escherichia coli, K.pneum - klebsiella pneumoniae, MRSA- methicillin resistant staph aureus, P.Aeru- pseudomonas aeruginosa, S.pneum- streptococcus pneumoniae)

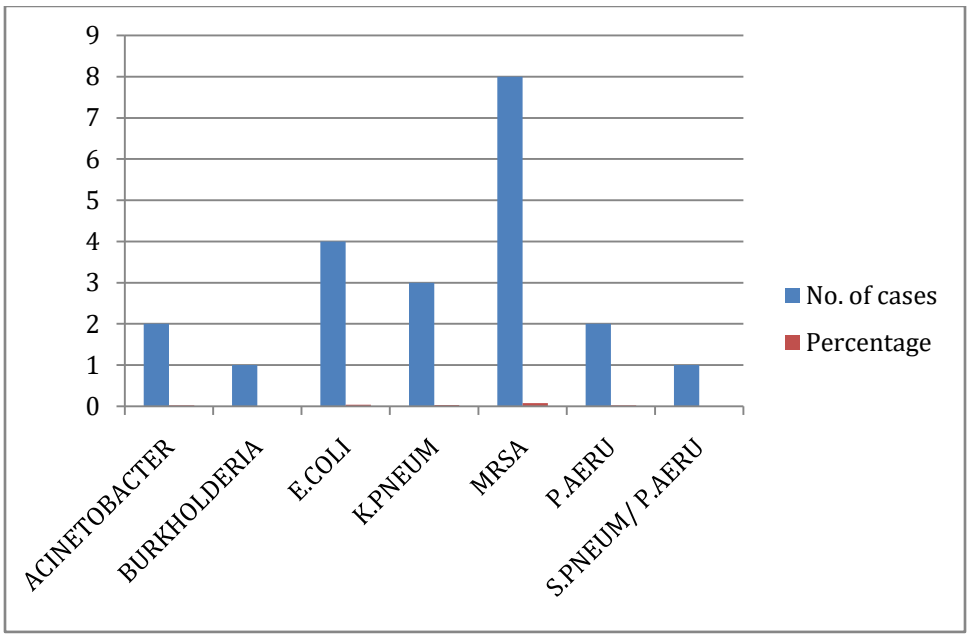

Figure 39 - Cases with positive culture sensitivity (e.coli- escherichia coli, K.pneum- klebsiella pneumoniae, MRSA- methicillin resistant staph aureus, P.Aeru- pseudomonas aeruginosa, S.pneum- streptococcus pneumoniae)

PF FINDING	NO OF CASES	PERCENTAGE (IN THEIR GROUP)
AFB	(n-42)2	4.76%
M CELLS	(n-9)7	77.77%

Table 42 - Other pleural fluid findings (AFB- acid fast bacilli, M cells- malignant cytology)

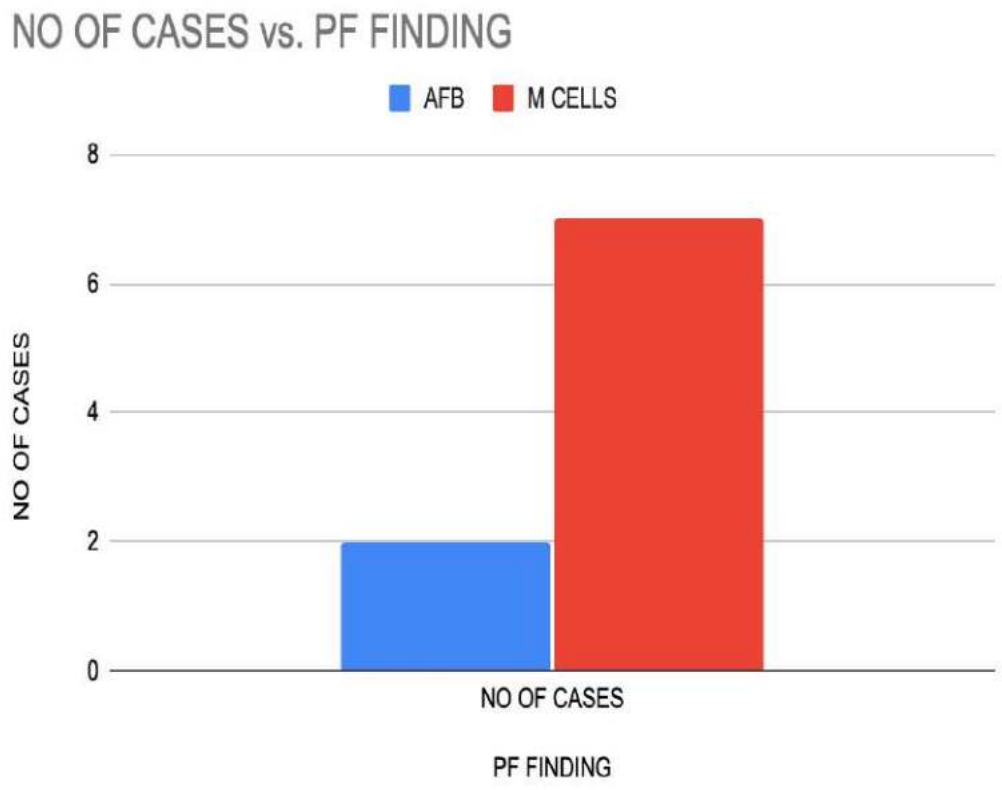

Figure 40 - Other pleural fluid findings (AFB- acid fast bacilli, M cells- malignant cytology)

Table 43- Association of pleural fluid M cells and malignant pleural effusion (M- malignant cytology)

		Malignant			Chi-square value	p-value
		Y	N	Total		
M CELL	NEG	2	91	93		
	POS	7	0	7		
Total		9	91	100	76.105	0.001

Table 44- Sensitivity and specificity of pleural fluid M cells and malignant pleural effusion (M- malignant cytology)

Statistic	Value	95% CI
Sensitivity	77.78%	39.99% to 97.19%
Specificity	100.00%	96.03% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	0.22	0.07 to 0.75
Disease prevalence (*)	9.00%	4.20% to 16.40%
Positive Predictive Value (*)	100.00%	
Negative Predictive Value (*)	97.85%	93.06% to 99.36%
Accuracy (*)	98.00%	92.96% to 99.76%

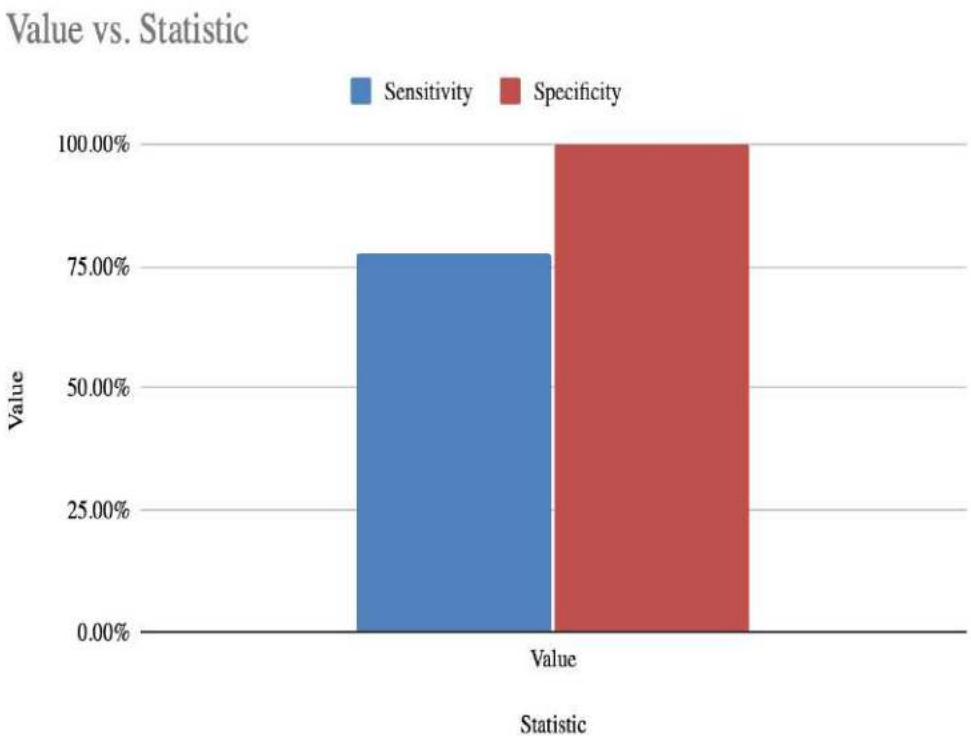

Figure 41- sensitivity and specificity of pleural fluid malignant cytology

OTHERS	NO.OF CASES	PERCENTAGE
SPUTUM E.COLI	1	1%
SPUTUM AFB/CBNAAT/MYCOBACTERIA	1	1%
SPUTUM K.PNEUM	1	1%
CA LUNG	7	7%
CA OVARY	1	1%
UNKNOWN PRIMARY	1	1%
HODGKIN'S LYMPHOMA	1	1%
MANTOUX	5	11.90%
TUMOR MARKERS	2	2%
LN- GRANULOMATOUS DISEASE	1	1.0%
LN- TB	1	1.0%
LUNG BX- ADENOCARCINOMA	4	4.0%
LUNG BX- NSCC	1	1%
P BX- CHRONIC INFLAMMATION	2	2.0%
COVID RTPCR POSITIVE	2	2.0%
E.HISTOLYTICA POSITIVE SEROLOGY	2	2.0%

Table 45 - Other relevant findings and figures (e.coli- escherichia coli, AFB- acid fast bacilli, CBNAAT- cartridge based nucleic acid amplification test, K.pneum- klebsiella pneumoniae, NSCC- non small cell carcinoma, CA- carcinoma, LN- lymph node, TB- tuberculosis, BX- Biopsy, E.histolytica- entameba histolytica, RT PCR- reverse transcriptase polymerase chain reaction)

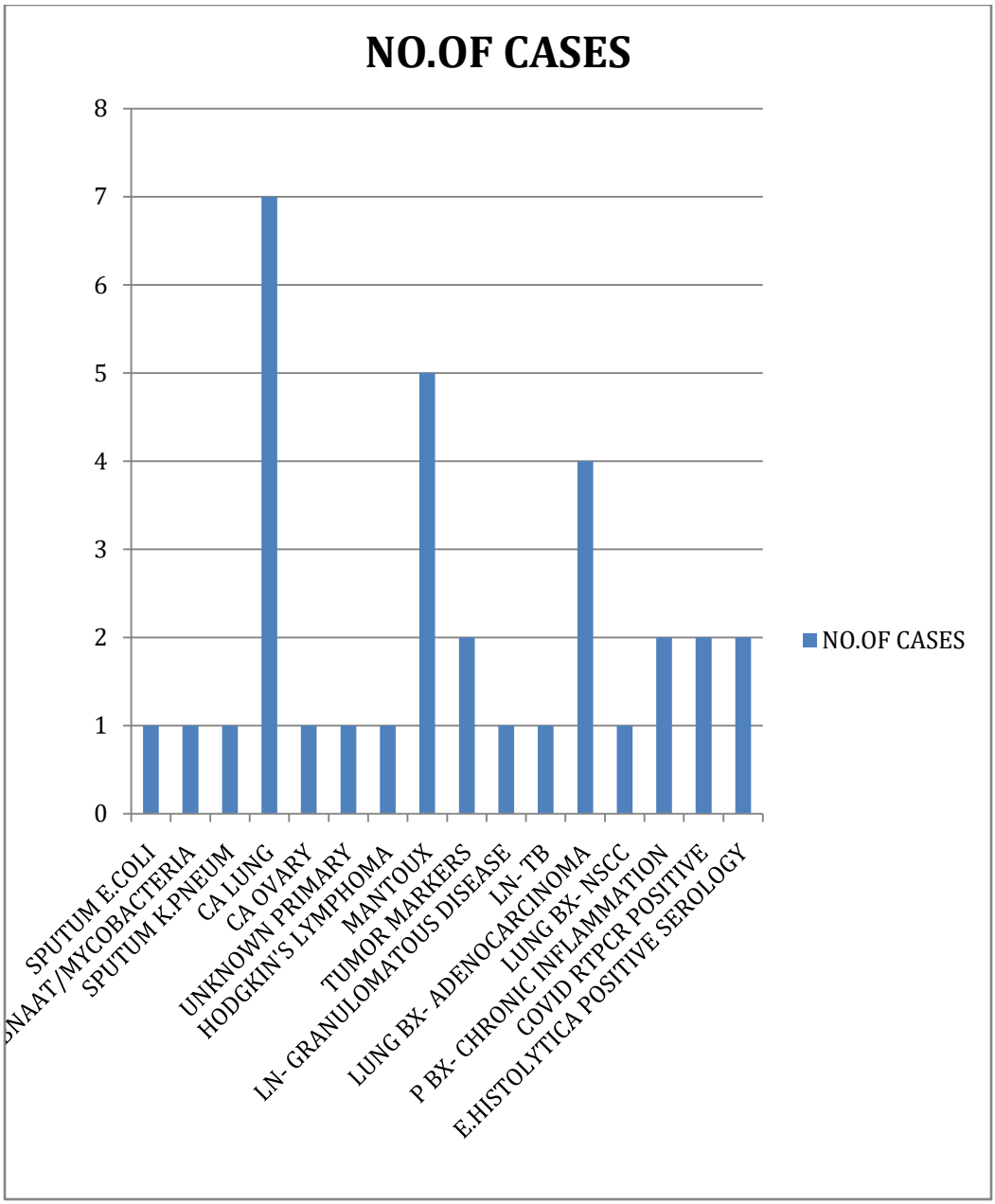

Figure 42 - Other relevant findings and figures (e.coli-escherichia coli, AFB-acid fast bacilli, CBNAAT-cartridge based nucleic acid amplification test, K. pneum-klebsiella pneumoniae, NSCC- non small cell carcinoma, CA-carcinoma, LN- lymph node, TB-tuberculosis, BX-Biopsy, E.histolytica-entameba histolytica, RT PCR-reverse transcriptase polymerase chain reaction)

Table 46 - Detailed gross appearance of "other' causes

	no. of patients (total)	Appearance				
		STRAW	HEMORRHAGIC	PUS	CHYLOUS	ANCHOVY SAUCE
PULMONARY EMBOLISM	3	1	2	0	0	0
HEMOTHORAX	3	0	3	0	0	0
PARASITIC	2	0	0	0	0	2
VIRAL	2	2	0	0	0	0
CHYLOUS	1	0	0	0	1	0

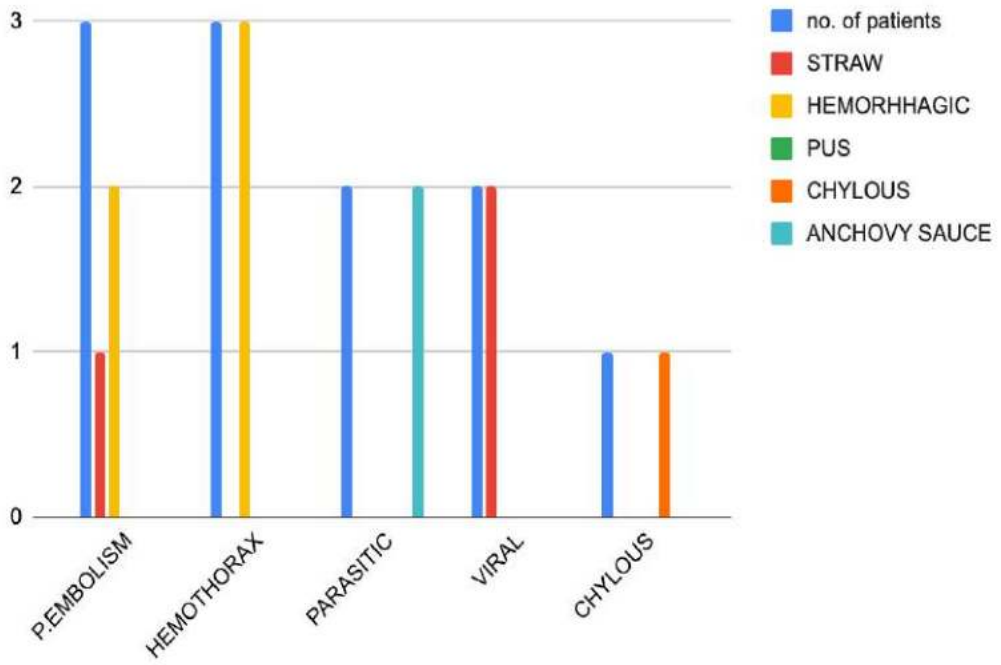

Figure 43- Detailed gross appearance of "other' causes (P.Embolism- Pulmonary Embolism)

Table 47- Detailed differential leukocyte count and biochemistry of pleural fluid with "other" causes

	TLC- (Mean) Cells/Mm^3	Lymphocyte Predominance (No. Of Cases)	Neutrophil Predominance (No. Of Cases)	Mean Protein (G/Dl)	Mean Ldh (U/L)
Pulmonary Embolism	166.6	2	1	3.93	286.6
Hemothorax	766.6	1	2	4.8	640
Parasitic	805	0	2	2.65	315
Viral	4000	1	1	2.85	845.5
Chylous	100	1	0	2.9	221

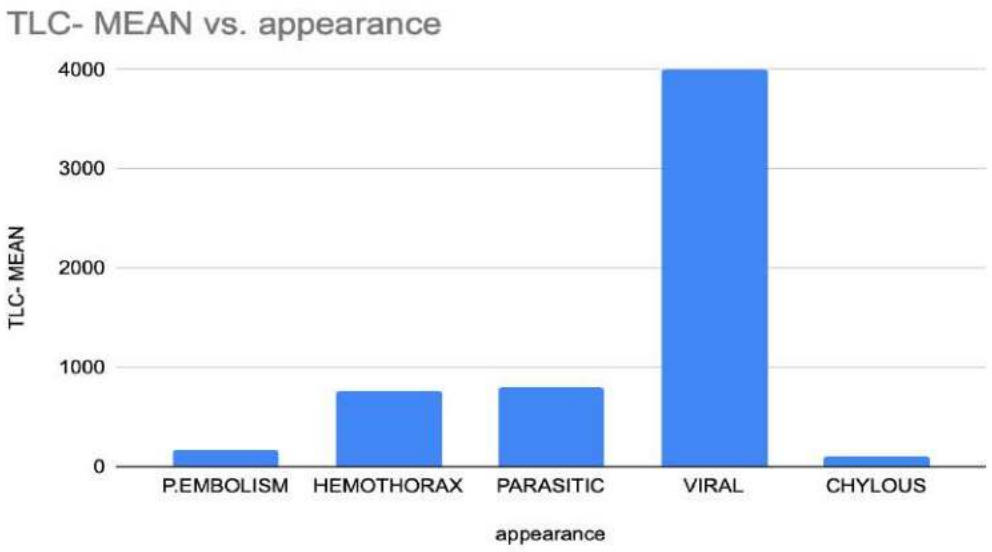

Figure 44- Detailed differential leukocyte count of 'other' causes (P.Embolism-Pulmonary Embolism)

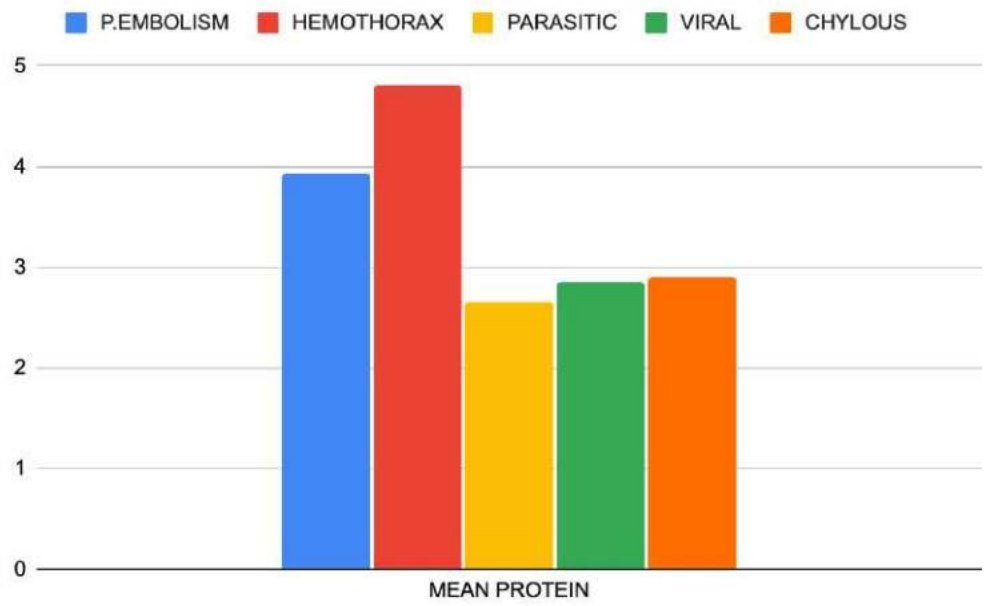

Figure 45- Detailed biochemistry (mean protein g/dl) of pleural fluid with 'other' causes (P.Embolism-Pulmonary Embolism)

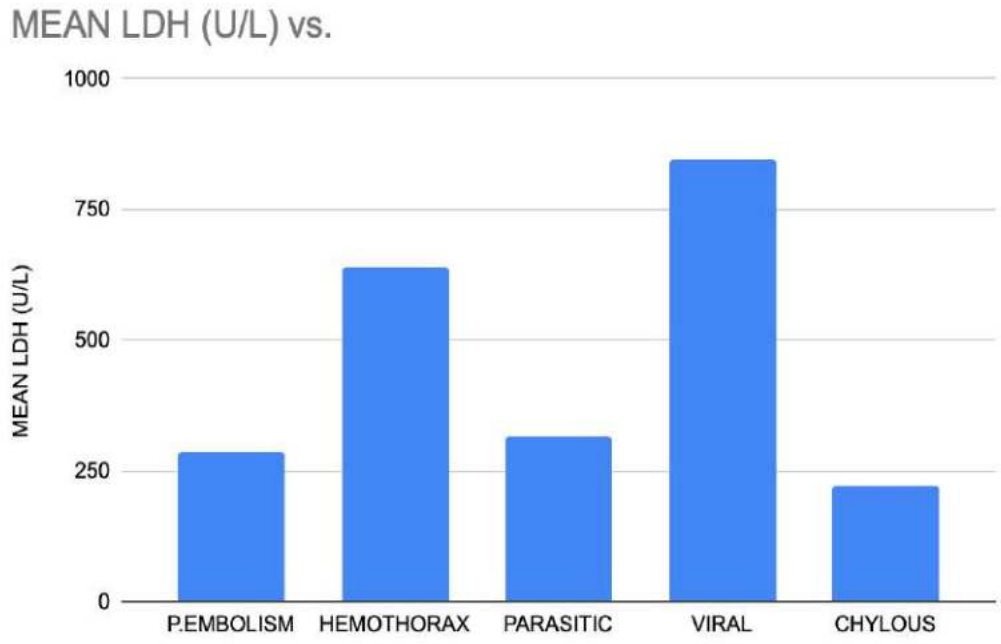

Figure 46 - Detailed biochemistry (mean LDH U/L) of pleural fluid with 'other' causes (P.Embolism-Pulmonary Embolism)

Table 48- Pleural fluid PCV in diagnosing hemothorax (PCV- Pack cell volume)

blood pcv vs pf pcv				
	Blood	**Pleural Fluid**	**Pleural fluid Pcv/ Blood Pcv**	**Hemothorax**
	29.8	15.8	0.53	y
	32	16.4	0.51	y
	39.2	20.2	0.51	y
PCV %	38.2	4	0.10	n

Table 49 - CT chest findings on 'other' causes (GGO- ground glass opacities)

	CT CHEST	
Etiology	**EMBOLISM**	**GGO**
PULMONARY EMBOLISM	3	0
HEMOTHORAX	0	0
PARASITIC	0	0
VIRAL	0	2
CHYLOUS	0	0

Table 50- Relevant test specific to etiology (E. Serology- entamoeba histolytica serology, TG- triglycerides)

OTHER TEST SPECIFIC TO ETIOLOGY			
ETIOLOGY	**RTPCR**	**E.SEROLOGY**	**TG >110 mg/dl**
PULMONARY EMBOLISM	0	0	0
HEMOTHORAX	0	0	0
PARASITIC	0	2	0
VIRAL	2	0	0
CHYLOUS	0	0	1

Table 51- Intervention and treatment in 'other' causes (PCD- percutaneous drainage, ICD- intercostal drainage, ABX- antibiotics, LMWH- low molecular weight heparin)

TREATMENT						
	Total	**PCD/ICD**	**ABX**	**LMWH**	**STEROIDS**	**ANTIVIRALS**
PULMONARY EMBOLISM	3	1	3	3	0	0
HEMOTHORAX	3	3	3	0	0	0
PARASITIC	2	2	2	0	0	0
VIRAL	2	0	2	0	2	2
CHYLOUS	1	1	1	0	0	0

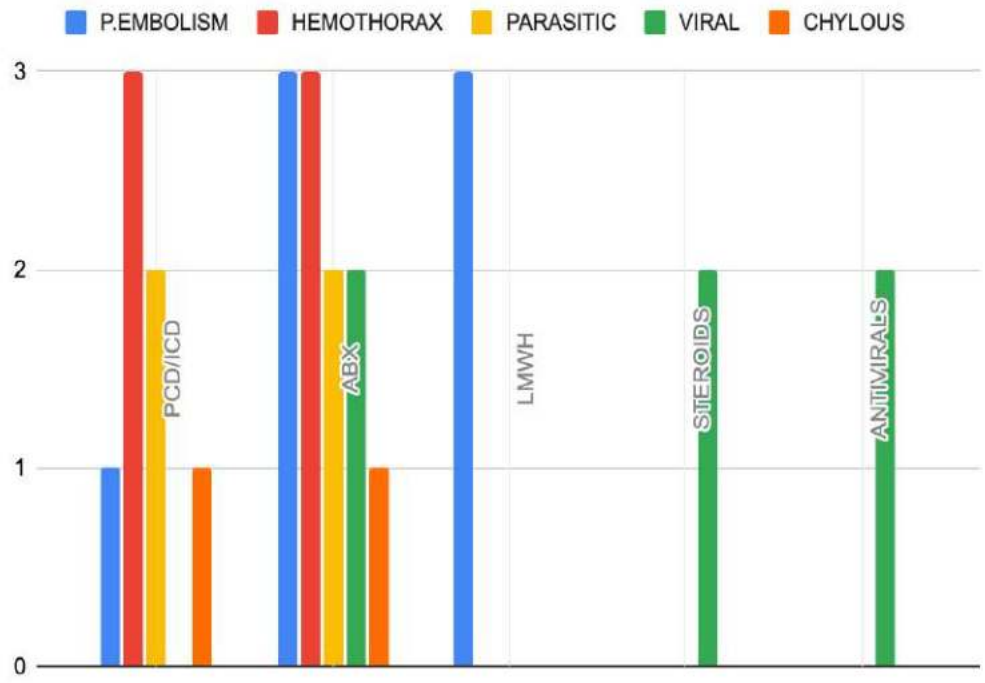

Figure 47- Intervention and treatment in 'other' causes (PCD- percutaneous drainage, ICD- intercostal drainage, ABX- antibiotics, LMWH- low molecular weight heparin, P.Embolism-Pulmonary Embolism)

Table 52- Outcome of patients with 'other' causes (DAMA- discharge against medical advice , EXP- expired, LTF- loss to follow up, RES- resolution)

		Outcome At D/C		Outcome At 1 Months			Outcome At 3 Months		
	Total	DAMA	EXP	EXP	LTF	RES	EXP	LTF	RES
PULMONARY EMBOLISM	3	0	0	0	0	3	0	0	3
HEMOTHORAX	3	0	1	1	0	2	1	0	2
PARASITIC	2	0	0	0	0	2	0	0	2
VIRAL	2	0	0	0	0	2	1	0	1
CHYLOUS	1	0	0	0	0	1	0	0	1

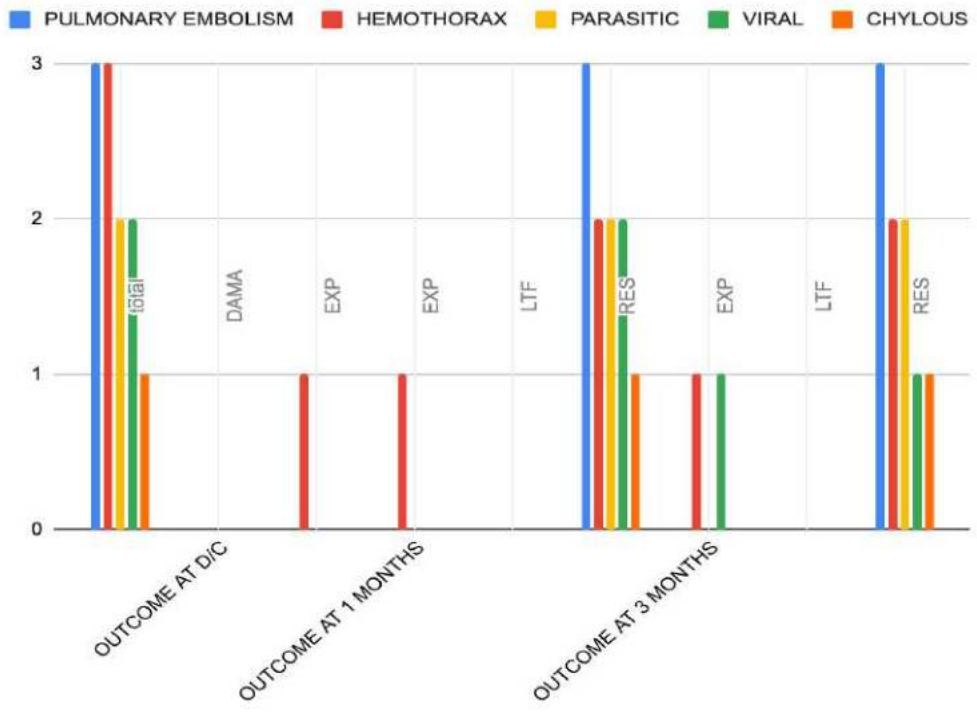

Figure 48 - Outcome of patients with 'other' causes (DAMA- discharge against medical advice, EXP- expired, LTF- loss to follow up, RES- resolution P.EMBOLISM-PULMONARY EMBOLISM)

Table 53- Follow up chest x ray of patients with 'other' causes (MOD- moderate, NA- not applicable, RES- resolved)

		CXR 1 MONTH					CXR AT 3 MONTH				
	Total	MILD	MOD	RES	PLEURA THICK	NA	MILD	MOD	RES	NA	PLEURA THICK
PULMONARY EMBOLISM	3	0	0	3	0	0	0	0	3	0	0
HEMOTHORAX	3	0	0	1	1	1	0	0	0	1	2
PARASITIC	2	0	0	2	0	0	0	0	2	0	0
VIRAL	2	2	0	0	0	0	1	0	0	1	0
CHYLOUS	1	1	0	0	0	0	1	0	0	0	0

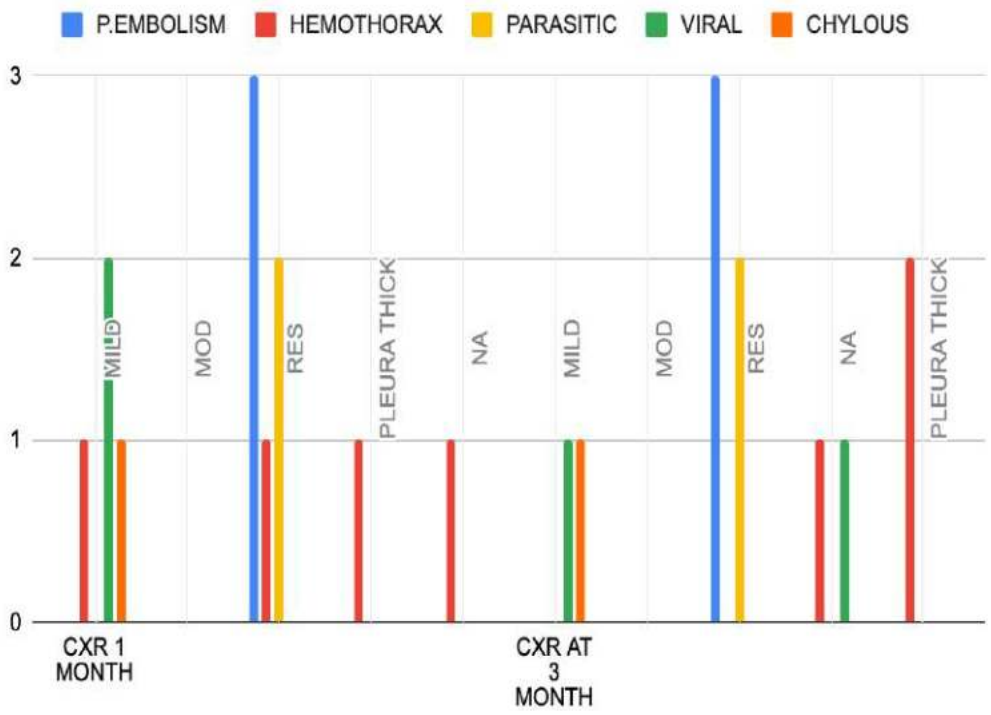

Figure 49 - Outcome of patients with 'other' causes (MOD- moderate, NA- not applicable, RES- resolved, P. embolism- Pulmonary embolism)

TEST	VALUES	NO OF CASES	PERCENTAGE
HB (G/DL)	<8	3	3.0%
	8-11.9	60	60.0%
	>12	37	37.0%
TLC (10^3/UL)	< 10	42	42.0%
	10-20.0	44	44.0%
	>20	14	14.0%
Creatinine >1.1MG/DL		35	35.0%
ESR> 40		84	84.0%
S. LDH >200U/L		42	42.0%
SGOT > 40U/L		58	58.0%
SGPT > 40U/L		59	59.0%

Table 54 - Relevant lab investigations (HB- hemoglobin, TLC- total leukocyte count, ESR- erythrocyte sedimentation rate, S.LDH- serum lactate dehydrogenase, SGOT- serum glutamic oxaloacetic transaminase, SGPT- serum glutamic pyruvic transaminase).

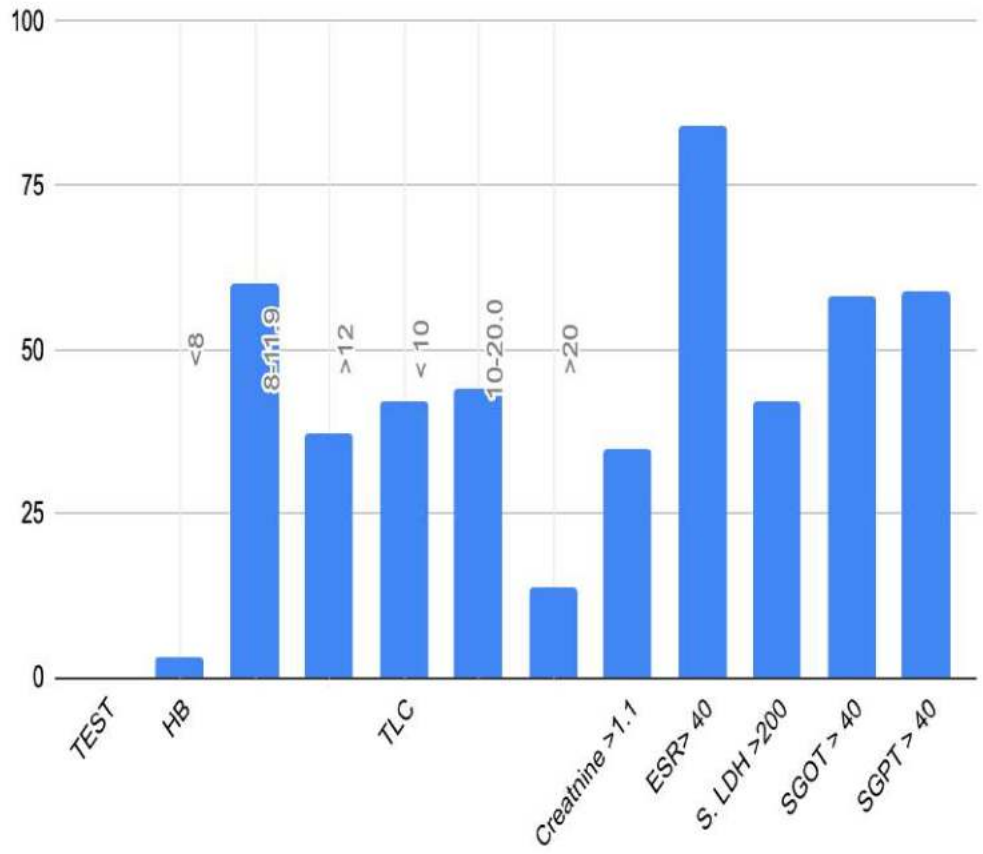

Figure 50 - Relevant lab investigations (HB- hemoglobin, TLC- total leukocyte count, ESR- erythrocyte sedimentation rate, S.LDH- serum lactate dehydrogenase, SGOT- serum glutamic oxaloacetic transaminase, SGPT- serum glutamic pyruvic transaminase).

	No. of cases	Percentage
TREATMENT ABX	64	64.0%
TREATMENT ATT	41	41.0%
TREATMENT CHEMO	7	7.0%
TREATMENT RADIO	0	0.0%
TREATMENT SX	3	3.0%

Table 55- Treatment received during hospitalization part a (ABX- antibiotic, ATT- antitubercular therapy, CHEMO- chemotherapy, RADIO- radiotherapy, SX- surgery)

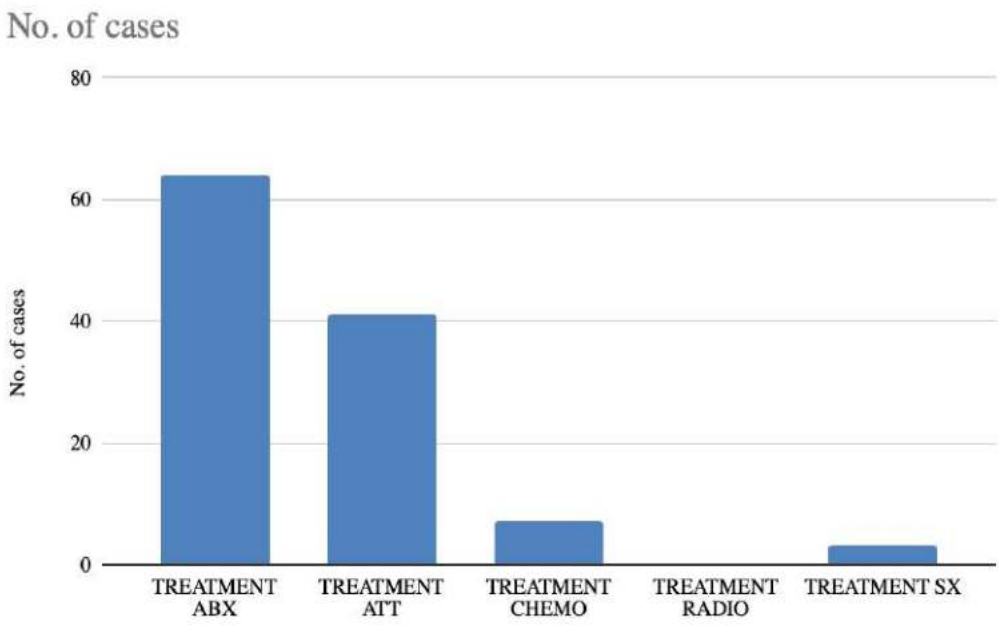

Figure 51 -Treatment received during hospitalization part a (ABX- antibiotic, ATT- antitubercular therapy, CHEMO- chemotherapy, RADIO- radiotherapy, SX- surgery)

TREATMENT OTHER	**No. of cases**	**Percentage**
INOTROPES	5	5.0%
LMWH	3	3.0%
MOD ATT	1	1.0%
STRD	5	5.0%
STRD + ANTIVIRAL	2	2.0%
Total	100	100.0%

Table 56 - Treatment received during hospitalization part b (LMWH- low molecular weight heparin, MOD ATT- Modified antitubercular therapy, STRD- STEROIDS)

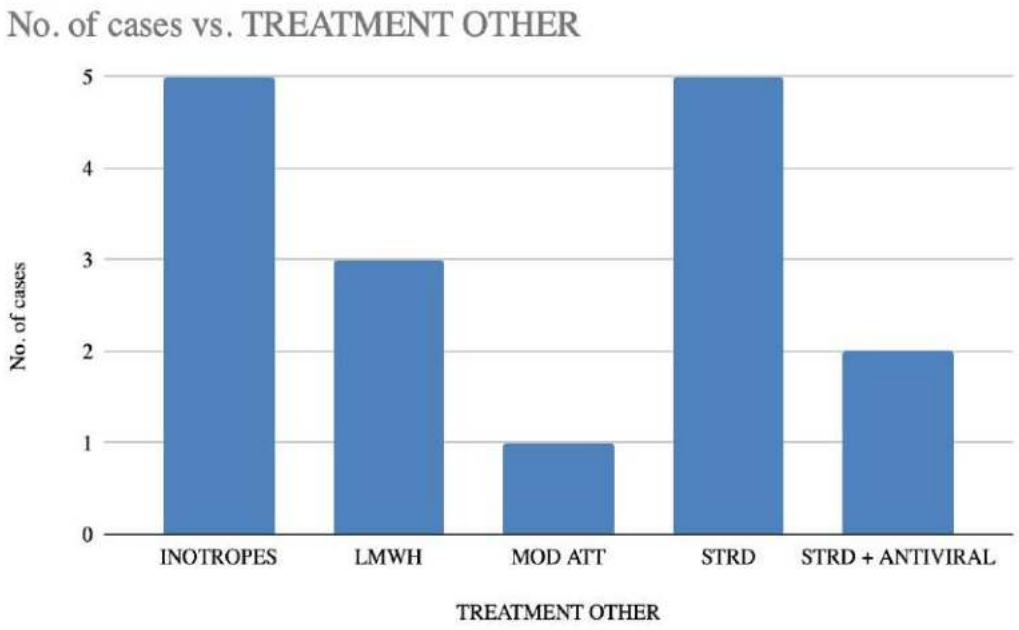

Figure 52 - Treatment received during hospitalization part b (LMWH- low molecular weight heparin, MOD ATT- Modified antitubercular therapy, STRD- STEROIDS)

INTERVENTION	No. of cases	Percentage
ICD	7	7.0%
ICD + STK	2	2.0%
N	50	50.0%
PCD	27	27.0%
PCD + STK	14	14.0%
Total	100	100.0%

Table 57- Distribution of patients who required intervention (ICD- intercostal drainage, STK- streptokinase, PCD- percutaneous drainage)

Table 58 - Distribution of patients who required intervention according to underlying etiology (ICD- intercostal drainage, STK- streptokinase, PCD- percutaneous drainage, TB- tuberculosis)

		INTERVENTION					Total
		ICD	ICD + STK	N	PCD	PCD + STK	
Etiology	TB	0	0	33	6	3	42
	Malignant	0	0	6	3	0	9
	Empyema	0	1	0	7	7	15
	Parapneumonic	3	1	1	7	4	16
	Others	4	0	4	3	0	11
	Unknown	0	0	6	1	0	7
Total		7	2	50	27	14	100

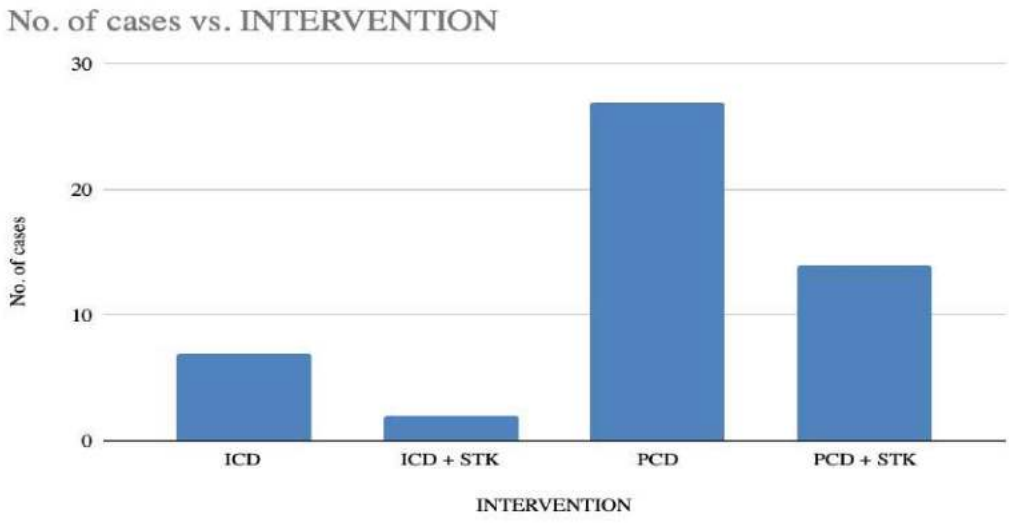

Figure 53 - Distribution of patients who required intervention (ICD- intercostal drainage, STK- streptokinase, PCD- percutaneous drainage)

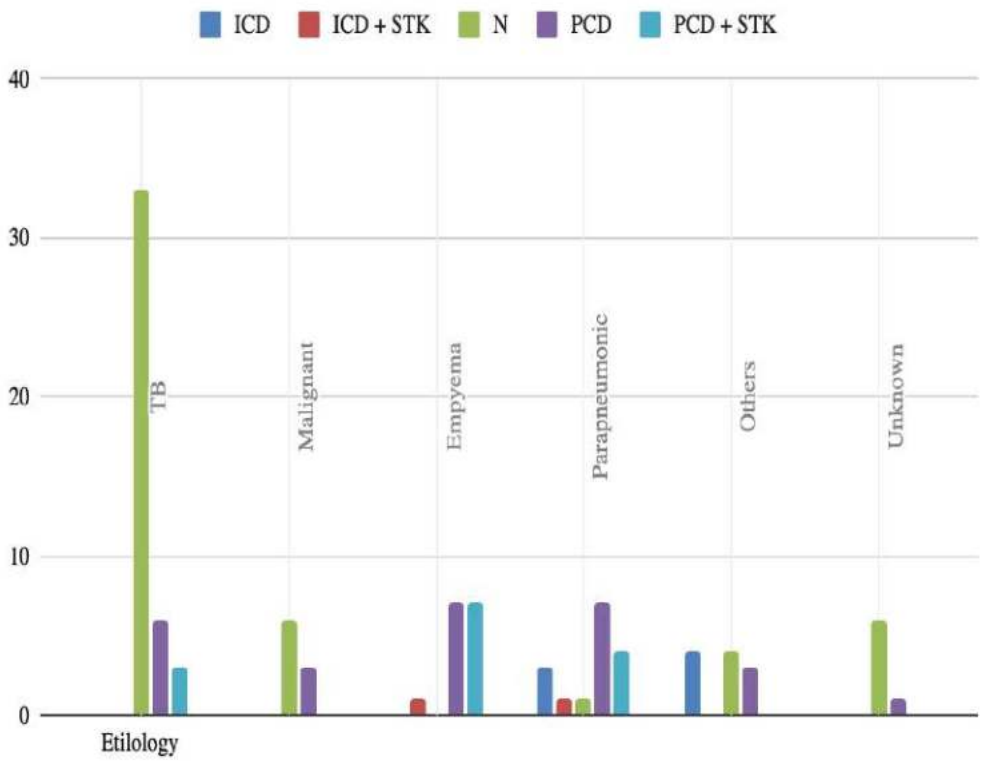

Figure 54 - Distribution of patients who required intervention according to underlying etiology (ICD- intercostal drainage, STK- streptokinase, PCD- percutaneous drainage, TB- tuberculosis).

		Etiology						
		TB	**malignant**	**empyema**	**parapneumonic**	**others**	**unknown**	**Total**
OUTCOME AT D/C	DAMA	0	2	0	1	0	0	3
	EXP	1	0	3	2	1	0	7
	NA	41	7	12	13	10	7	90
OUTCOME 1 MONTH	EXP	1	2	4	3	1	0	11
	LTF	0	1	0	0	0	0	1
	NA	41	6	11	13	10	7	88
OUTCOME 3 MONTH	EXP	1	3	4	3	2	0	13
	LTF	0	2	0	0	0	0	2
	NA	41	4	11	13	9	7	85
Total		42	9	15	16	11	7	100

Table 59- Outcome of patients according to etiology (TB, tuberculosis, DAMA- discharge against medical advice, EXP- expired, NA- not applicable, LTF- lost to follow up)

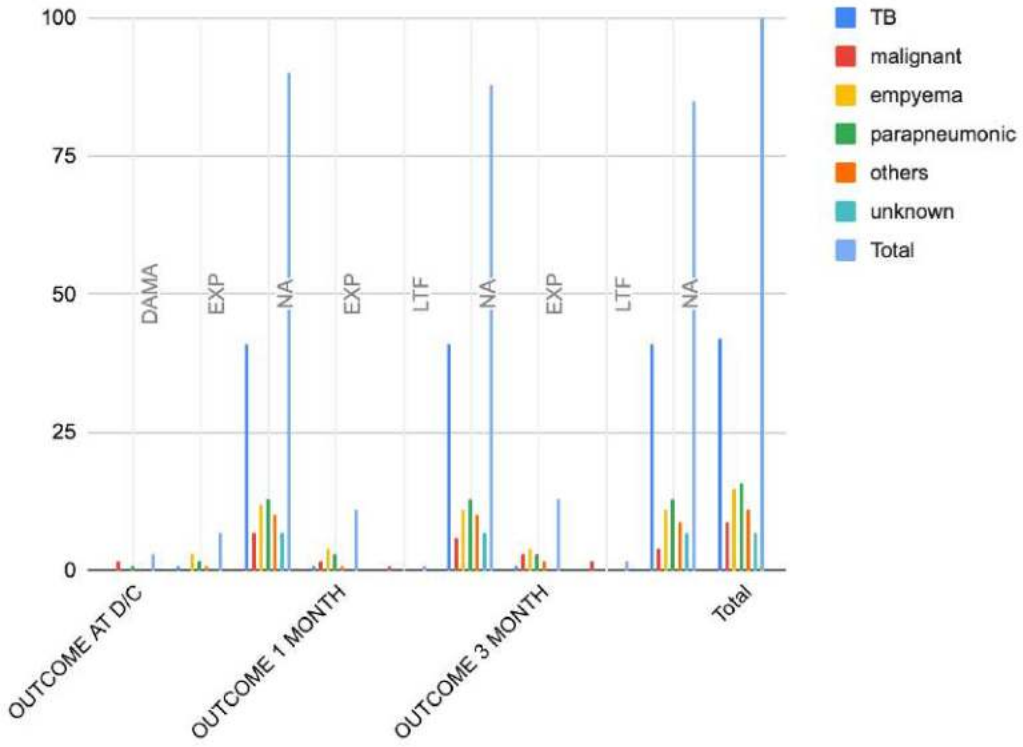

Figure 55- Outcome of patients according to etiology (TB, tuberculosis, DAMA- discharge against medical advice, EXP- expired, NA- not applicable, LTF- lost to follow up)

Table 60- Follow up chest x ray according to underlying etiology (MOD- moderate, PCD- percutaneous drainage, RES- resolution, SIGNFCNT- significant, P thick- pleural thickening, NA- not applicable)

		Etiology						
		tb	malignant	empyema	parapneumonic	others	unknown	Total
CHEST X RAY AT DISCHARGE	MILD	21	3	4	6	9	6	49
	MOD	13	4	2	3	0	1	23
	NA	1	1	3	2	1	0	8
	PCD IN SITU	6	0	3	4	0	0	13
	RES	1	0	3	1	1	0	6
	SIGNFCNT	0	1	0	0	0	0	1
CHEST X RAY AT 1 MONTH	MILD	18	4	3	6	4	1	36
	MOD	0	2	1	1	0	1	5
	NA	1	3	4	3	1	0	12
	P THICK	4	0	0	1	1	1	7
	RECURRENCE	1	0	0	0	0	3	4
	RES	18	0	7	5	5	1	36
CHEST XRAY AT 3 MONTH	MILD	6	2	1	4	2	0	14
	MOD	0	0	0	1	0	1	2
	NA	1	5	4	4	2	0	13

	P THICK	7	0	1	1	2	2	13
	RECURRENCE	1	2	0	0	0	3	6
	RES	27	0	9	6	5	1	49
Total		42	9	15	16	11	7	100

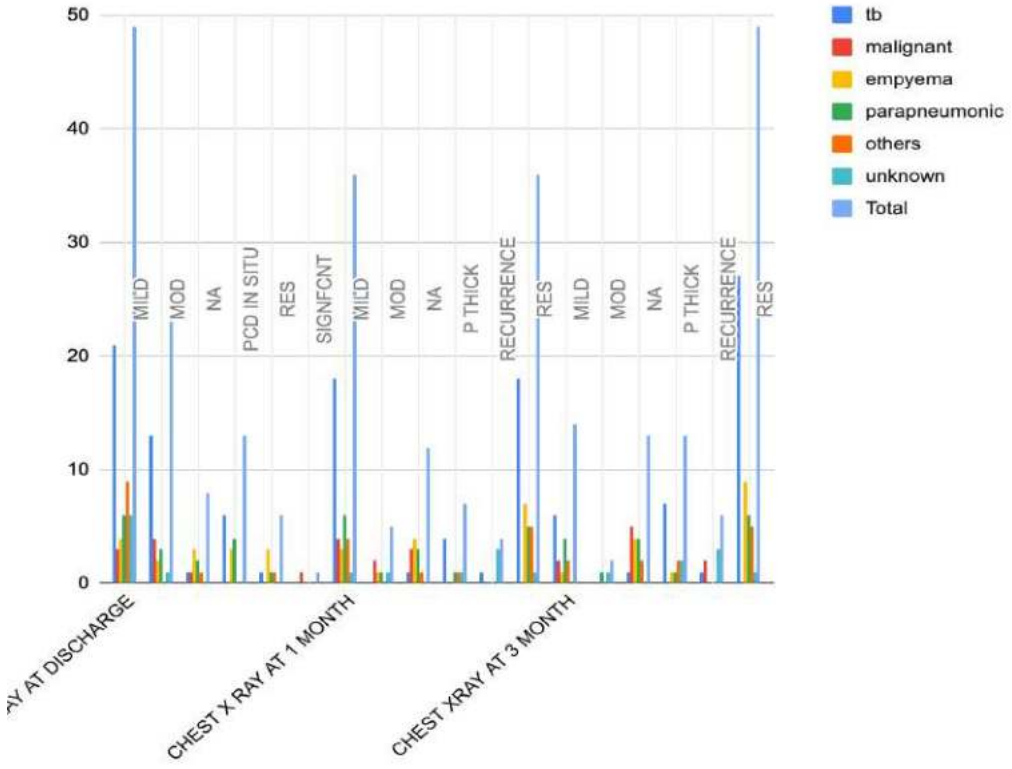

Figure 56 - Follow up chest x ray according to underlying etiology (MOD- moderate, PCD- percutaneous drainage, RES- resolution, SIGNFCNT- significant, P thick- pleural thickening, NA- not applicable)

		Etiology						
		tb	malignant	empyema	parapneumonic	others	unknown	Total
F/U CT CHEST 1 MONTH	Antr. Mediastinal Mass-Lymphoma	1	0	0	0	0	0	1
	Lt.Mod P.E	0	0	0	1	0	0	1
	Lymphangitis Carcinomatosis	0	0	0	0	0	1	1
	N	40	6	12	12	10	6	86
	Na	1	3	3	3	1	0	11
F/U CT CHEST 3 MONTHS	Mod Lt P.E	0	0	0	0	0	1	1
	N	41	4	11	11	9	7	83
	Na	1	5	4	1	2	0	16

Table 61- Follow up CT chest according to etiology (LT- left, MOD- moderate, P.E- pleural effusion, N- no, NA- not applicable, tb- Tuberculosis, F/U-follow up)

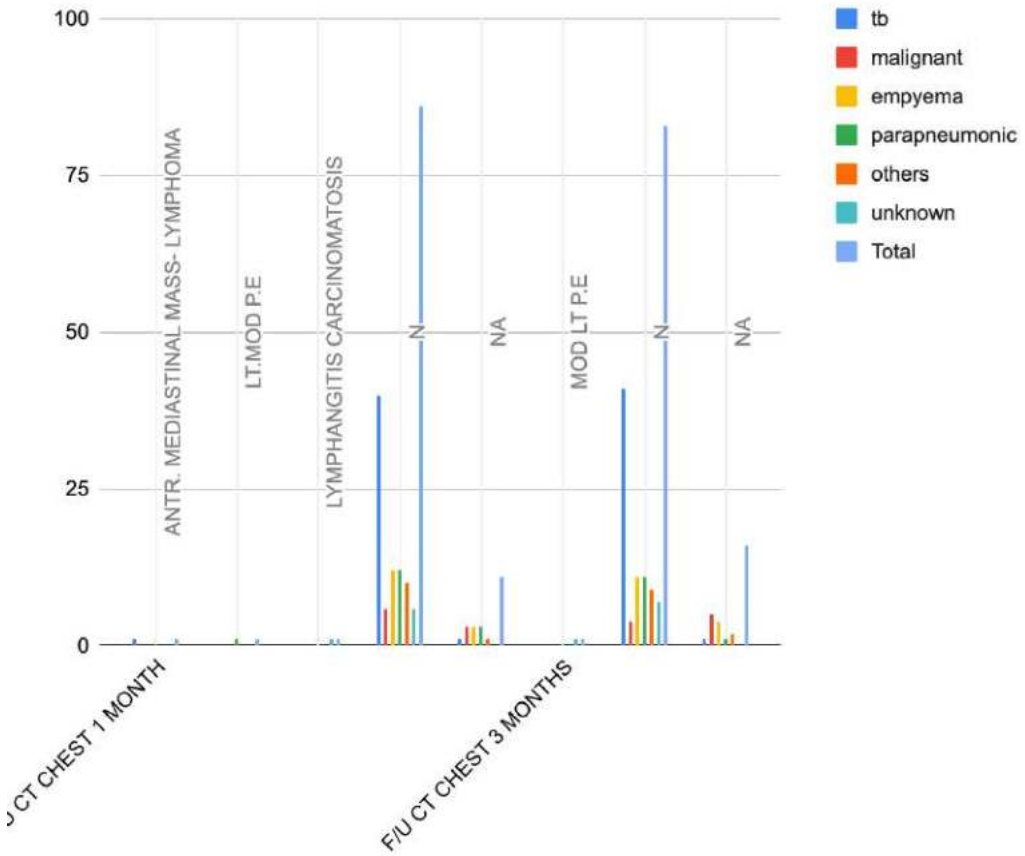

Figure 57 - Follow up CT chest according to etiology (LT- left, MOD- moderate, P.E- pleural effusion, N- no, NA- not applicable, tb- Tuberculosis).

		Etiology						
		tb	**m**	**empyema**	**parapneumonic**	**others**	**unknown**	**Total**
CHANGE IN DIAGNOSIS AT 1 MONTH	M.P.E	1	0	0	0	0	1	2
	N	40	6	11	12	10	2	81
	NA	1	3	4	3	1	0	12
	UNKNOWN	0	0	0	1	0	4	5
CHANGE IN DIAGNOSIS AT 3 MONTHS	N	40	6	11	11	9	7	84
	NA	1	5	4	4	2	0	16
Total		41	11	16	15	10	7	100

Table 62- Any change in diagnosis on follow up of the patients (M.P.E- malignant pleural effusion, N- no, NA- not applicable)

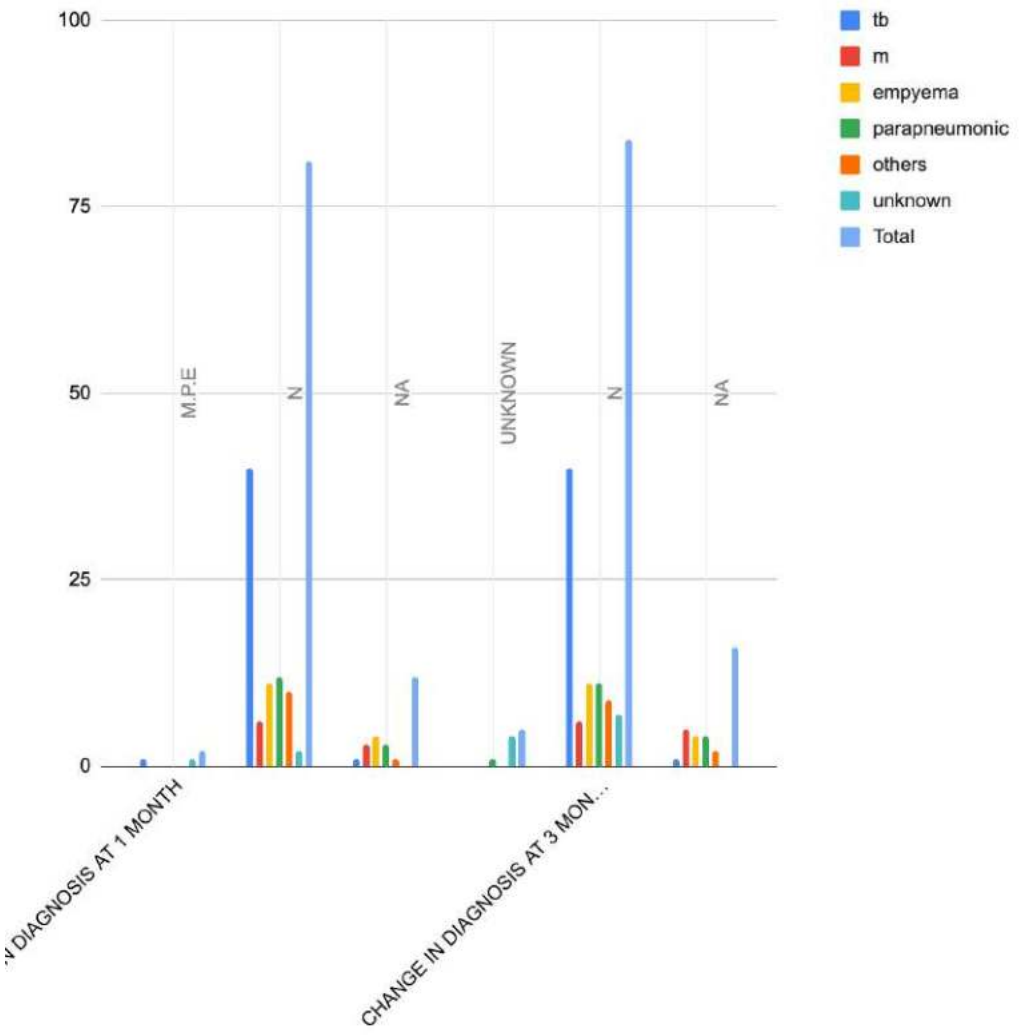

Figure 58 - Any change in diagnosis on follow up of the patients. (M.P.E- malignant pleural effusion, N- no, NA- not applicable).

		Etiology						
		TB	**M**	**E**	**PP**	**OTHERS**	**UNKNOWN**	**Total**
COMPLICATION AT DISCHARGE	H.PNEUMOTHORAX	1	0	0	0	0	0	1
	N	40	7	11	13	10	7	88
	NA	1	2	4	3	1	0	11
COMPLICATION 1 MONTH	AKI	1	0	0	0	0	0	1
	CHEST PAIN	1	0	0	0	0	0	1
	HEPATITIS	7	0	0	0	0	0	7
	N	31	6	11	13	10	4	75
	NA	1	3	4	3	1	0	11
	RECURRENCE	1	0	0	0	0	3	4
COMPLICATION 3 MONTH	CHEST PAIN	1	0	0	0	0	0	1
	COVID POSITIVE - EXP	0	1	0	0	0	0	1
	METS TO BRAIN	0	1	0	0	0	0	1
	N	39	3	11	12	9	5	79
	NA	1	4	4	4	2	0	15
	RECURRENCE	1	0	0	0	0	2	3
Total		42	9	16	16	10	7	100

Table 63- Distribution of complications at discharge and follow up (h.pneumothorax- hydropneumothorax, N- no-, NA- not applicable, AKI- Acute kidney injury, EXP- expired, METS- metastasis)

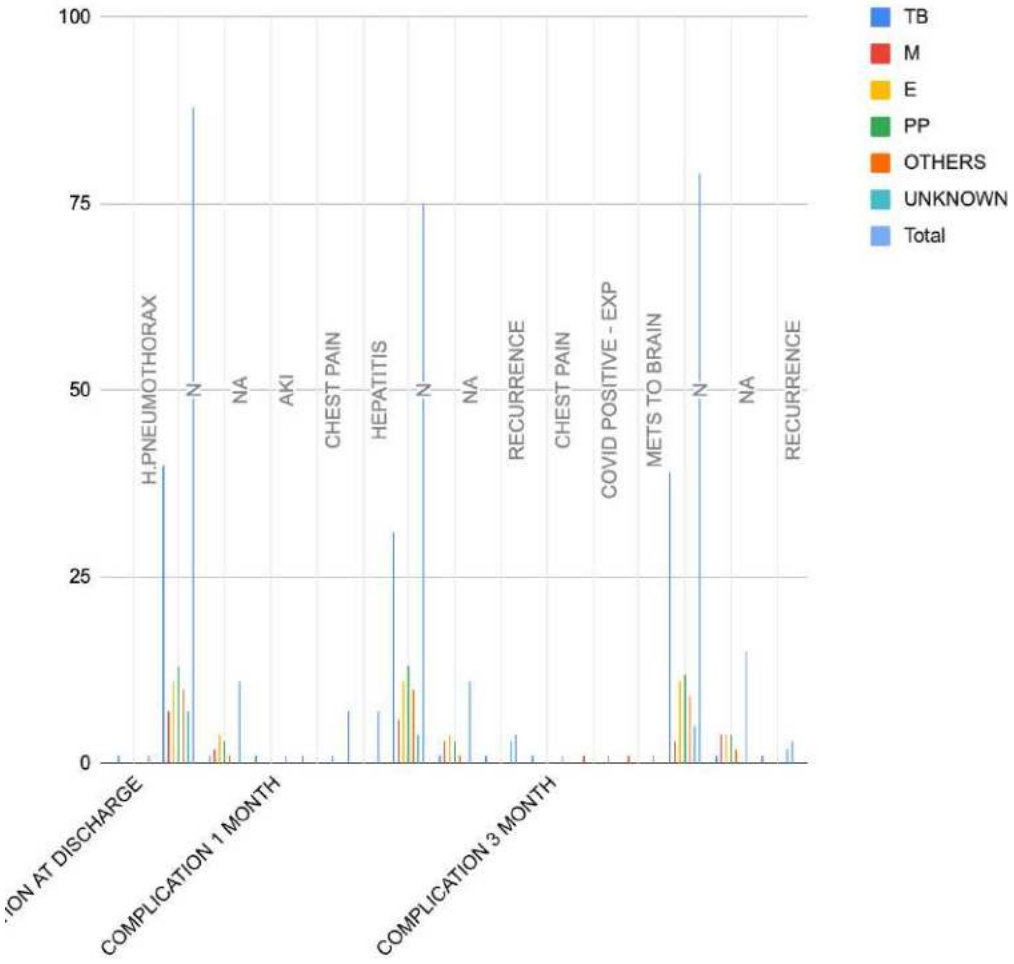

Figure 59- Distribution of complications at discharge and follow up (h.pneumothorax- hydropneumothorax, N- no-, NA- not applicable,AKI- Acute kidney injury, EXP- expired, METS- metastasis)

		Etiology						
		tb	**malignancy**	**empyema**	**parapneumonic**	**others**	**Unknown**	**Total**
TREATMENT CHANGE 1 MONTH	CHEMOTHERAPY	1	0	0	0	0	1	2
	MOD ATT	8	0	0	0	0	0	8
	N	30	6	12	13	10	4	75
	NA	1	3	3	3	1	0	11
	PCD OUT	2	0	0	0	0	0	2
	TH. P. TAP	0	0	0	0	0	1	1
	VATS	0	0	0	0	0	1	1
TREATMENT CHANGE AT 3 MONTH	N	41	2	10	12	9	5	79
	NA	1	5	4	4	2	0	16
	PCD	0	0	0	0	0	1	1
	PCD OUT	0	0	1	0	0	0	1
	TH. P. TAP	0	2	0	0	0	0	2
	VATS	0	0	0	0	0	1	1
Total		42	9	16	16	10	7	100

Table 64- Any change in treatment on follow up (mod att- modified antitubercular therapy, NA- not applicable, N- no, PCD- percutaneous drainage, TH. P. TAP- therapeutic pleural tap, VATS- video assisted thoracoscopy)

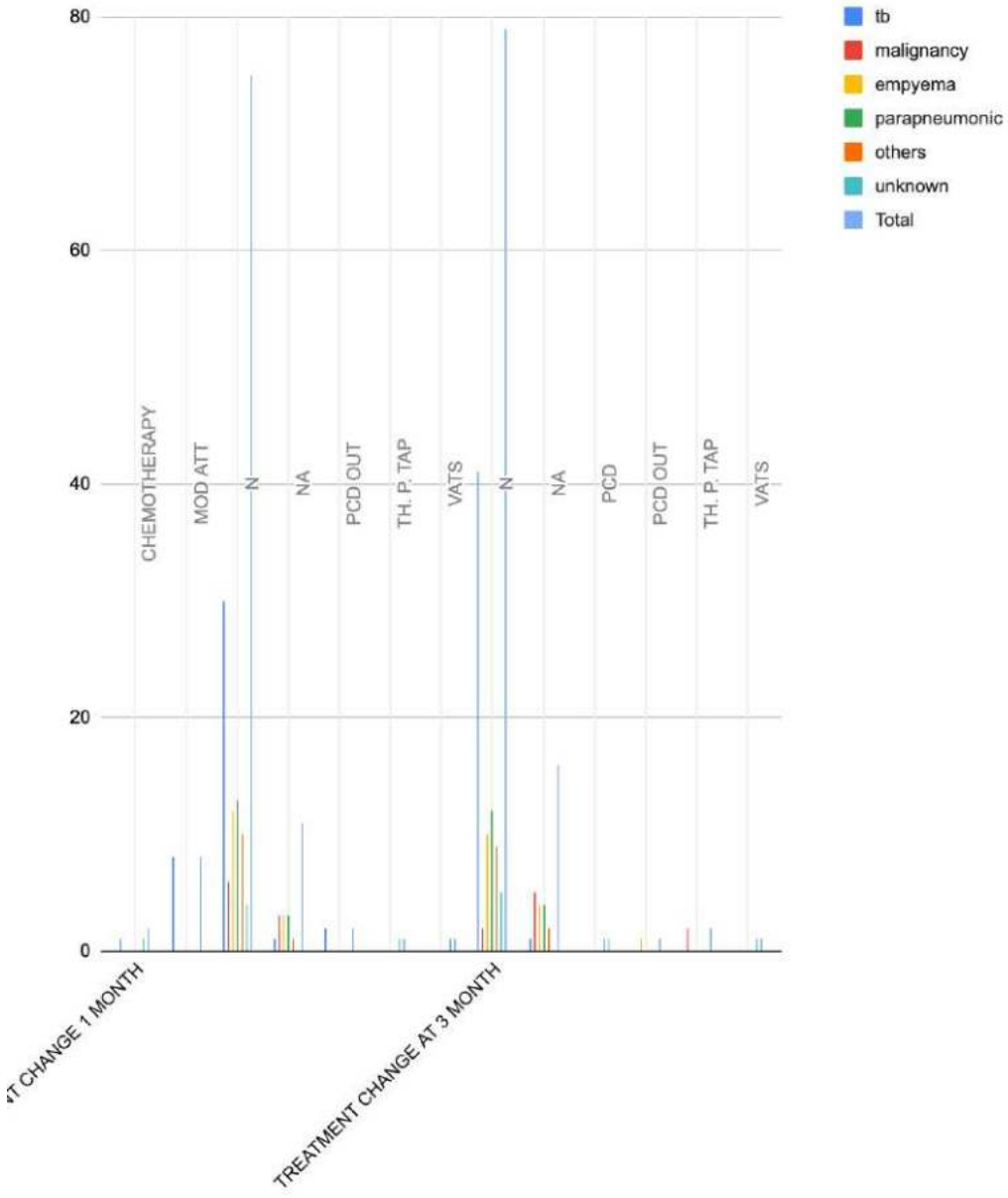

Figure 60- Any change in treatment on follow up (mod att- modified antitubercular therapy, NA- not applicable, N- no, PCD- percutaneous drainage, TH. P. TAP- therapeutic pleural tap, VATS- video assisted thoracoscopy)

		OUTCOME 3 MONTH				Total	Chi-square value	p-value
		Expired		Survival				
Age group (years)	< 20	0	0.0%	1	1.2%	1	3.063	0.382
	21-40	2	15.4%	31	36.5%	33		
	41-60	5	38.5%	30	35.3%	35		
	> 60	6	46.2%	23	27.1%	29		
GENDER	F	7	53.8%	19	22.4%	26	5.737	0.037
	M	6	46.2%	66	77.6%	72		
PRESENT HISTORY _CHEST PAIN	N	8	61.5%	42	49.4%	50	0.664	0.554
	Y	5	38.5%	43	50.6%	48		
SOB	N	2	15.4%	21	24.7%	23	0.545	0.46
	Y	11	84.6%	64	75.3%	75		
FEVER	N	3	23.1%	36	42.4%	39	0.749	0.183
	Y	10	76.9%	49	57.6%	59		
TRAUMA	N	11	84.6%	77	90.6%	88	0.439	0.508
	Y	2	15.4%	8	9.4%	10		
COUGH	N	5	38.5%	41	48.2%	46	0.432	0.511
	Y	8	61.5%	44	51.8%	52		

EXPECTORATION	N	7	53.8%	73	85.9%	80	7.718	0.013
	Y	6	46.2%	12	14.1%	18		
HEMOPTYSIS	N	13	100.0%	81	95.3%	94	0.6378	0.425
	Y	0	0.0%	4	4.7%	4		
PAST HSITORY_TB	N	11	84.6%	77	90.6%	88	0.439	0.508
	Y	2	15.4%	8	9.4%	10		
HTN	N	9	69.2%	67	78.8%	76	0.596	0.440
	Y	4	30.8%	18	21.2%	22		
DM	N	7	53.8%	69	81.2%	76	4.838	0.067
	Y	6	46.2%	16	18.8%	22		
CLD	N	12	92.3%	80	94.1%	92	0.064	0.585
	Y	1	7.7%	5	5.9%	6		
CKD	N	13	100.0%	83	97.6%	96	0.312	0.576
	Y	0	0.0%	2	2.4%	2		
PLHIV	N	13	100.0%	84	98.8%	97	0.155	0.694
	Y	0	0.0%	1	1.2%	1		
HCV	N	12	92.3%	83	97.6%	95	1.083	0.298
	Y	1	7.7%	2	2.4%	3		
HBV	N	13	100.0%	84	98.8%	97	0.155	0.694

	Y	0	0.0%	1	1.2%	1		
OTHER	BA	1	7.7%	0	0.0%	1	16.73	0.053
	CA LUNG	1	7.7%	1	1.2%	2		
	CA OVARY	1	7.7%	0	0.0%	1		
	CAD	0	0.0%	2	2.4%	2		
	COVID POSITIVE	0	0.0%	1	1.2%	1		
	HL	0	0.0%	1	1.2%	1		
	HYPOTHYROIDISM	0	0.0%	1	1.2%	1		
	HYSTERECTOMY	0	0.0%	1	1.2%	1		
	ON VENTILATORY SUPPORT	0	0.0%	1	1.2%	1		
BMI GROUP	< 18.9	1	7.7%	0	0.0%	1	6.804	0.078
	19-22.9	5	38.5%	38	44.7%	43		
	23-24.9	3	23.1%	24	28.2%	27		

	> 25	4	30.8%	23	27.1%	27		
SPO2 LESS THAN 94 REQUIRING OXYGEN	N	1	7.7%	63	74.1%	64	21.958	0.001
	Y	12	92.3%	22	25.9%	34		
ICTERUS	N	11	84.6%	84	98.8%	95	7.670	0.045
	Y	2	15.4%	1	1.2%	3		
CLUBBING	N	13	100.0%	83	97.6%	96	0.312	0.576
	Y	0	0.0%	2	2.4%	2		
LAP	N	13	100.0%	82	96.5%	95	0.473	0.491
	Y	0	0.0%	3	3.5%	3		
CVS	MUFFLED HEART SOUND	1	7.7%	0	0.0%	1	6.606	0.133
	NAD	12	92.3%	85	100.0%	97		
CNS	ENCEPHALOPATHY	5	38.5%	2	2.4%	7	22.165	0.0001
	NAD	8	61.5%	83	97.6%	91		
ABD	ASCITIS	3	23.1%	4	4.7%	7	5.844	0.054
	NAD	10	76.9%	80	94.1%	90		

	TENDER RIGHT UPPER QUADRANT	0	0.0%	1	1.2%	1		
Hb group (g/dl)	< 8	1	7.7%	2	2.4%	3	1.258	0.533
	8-11.9	8	61.5%	50	58.8%	58		
	> 12	4	30.8%	33	38.8%	37		
tlc group 10^3/UL	< 10	1	7.7%	41	48.2%	42	15.018	0.001
	10-20.0	6	46.2%	36	42.4%	42		
	> 20	6	46.2%	8	9.4%	14		
CREATININE group (mg/dl)	1.1	3	23.1%	60	70.6%	63	11.086	0.001
	<1.1	10	76.9%	25	29.4%	35		
ALBUMIN G/DL	>3.5	1	7.7%	37	43.5%	38	6.100	0.014
	<3.5	12	92.3%	48	56.5%	60		
ot U/L	<40	3	23.1%	38	44.7%	41	2.168	0.227
	>40	10	76.9%	47	55.3%	57		
pt U/L	<40	2	15.4%	38	44.7%	40	4.013	0.068
	>40	11	84.6%	47	55.3%	58		
S.LDH U/L	<200	2	15.4%	50	58.8%	52	8.819	0.003
	>200	11	84.6%	34	40.0%	45		

LATERALITY	BILATERAL	2	15.4%	5	5.9%	7	1.888	0.389
	LEFT	4	30.8%	37	43.5%	41		
	RIGHT	7	53.8%	43	50.6%	50		
MILD	N	13	100.0%	64	75.3%	77	4.088	0.064
	Y	0	0.0%	21	24.7%	21		
MODERATE	N	2	15.4%	38	44.7%	40	4.013	0.068
	Y	11	84.6%	47	55.3%	58		
SIGNIFICANT	N	11	84.6%	68	80.0%	79	0.154	0.695
	Y	2	15.4%	17	20.0%	19		
SEPTAE/ MULTIPLE LOCULATION	N	8	61.5%	73	85.9%	81	4.661	0.046
	Y	5	38.5%	12	14.1%	17		
LOCULATION-SINGLE	N	11	84.6%	74	87.1%	85	0.059	0.809
	Y	2	15.4%	11	12.9%	13		
PLEURAL FLUID GLUCOSE GROUP (mg/dl)	< 40	8	61.5%	19	22.4%	27	11.449	0.003
	40-100	2	15.4%	54	63.5%	56		
	>100	3	23.1%	12	14.1%	15		
TLC group (cells/mm3)	< 2500	11	84.6%	79	92.9%	90	2.491	0.288
	2500-5000	1	7.7%	5	5.9%	6		

	>5000	1	7.7%	1	1.2%	2		
Total		13	100.0%	85	100.0%	98		

Table- 65 Comparison of variables between expired and survival group (N- no, Y- yes, F- Female, M- male, SOB- shortness of breath, TB- tuberculosis, HTN- hypertension, DM- diabetes mellitus, CLD- chronic liver disease, CKD- chronic kidney disease, PLHIV- people living with human immunodeficiency virus, HBV- hepatitis b virus, HCV- hepatitis c virus, BA- bronchial asthma, CAD- Coronary artery disease, CA- carcinoma BMI- body mass index, spo_2 - oxygen saturation, LAP- lymphadenopathy, CVS- cardiovascular system, CNS- central nervous system, ABD- abdomen, HB- hemoglobin , TLC - total leukocyte count, OT- serum glutamic oxaloacetic transaminase, PT- serum glutamic pyruvic transaminase, NAD- no abnormality detected)

	Expired		Survival		z	p-value
	Mean	**Sd**	**Mean**	**Sd**		
AGE	57.62	12.67	47.80	18.41	1.853	0.067
HB	10.67	2.21	11.39	1.61	-1.433	0.155
TLC	23.33	10.02	11.31	5.71	6.297	0.000
CREATININE	2.58	1.70	1.17	1.51	3.080	0.003
PROTEIN	5.90	0.99	6.26	1.00	-1.219	0.226
OT	85.54	52.53	52.92	35.78	2.862	0.005
PT	75.46	48.48	49.96	36.53	2.240	0.027
S .LDH	316.62	127.98	222.05	118.24	2.655	0.009
pf PROTEIN	4.33	0.99	4.17	1.04	0.513	0.609
GLUCOSE	50.08	57.94	74.76	47.41	-1.697	0.093
pf LDH	932.38	514.03	656.12	798.75	1.206	0.231
pf TLC	1460.77	1962.57	808.35	1319.79	1.547	0.125

Table 66- Comparison of variables (average) between expired and survival group (,HB- hemoglobin, TLC - total leukocyte count, OT- serum glutamic oxaloacetic transaminase, PT- serum glutamic pyruvic transaminase, LDH-lactate dehydrogenase, pf- pleural fluid, NAD- no abnormality detected)

DISCUSSION

A prospective and observational study of 100 admitted patients with age more than 18 years who were having exudative pleural effusion by light's criteria was conducted in Dayanand medical college and hospital.

Of the 100 patients enrolled 27% were females and 73 % were males. In a similar study conducted by Gupta R et al.,[35] 75% of the patients were male and 25% of the patients were female. Another study conducted by Saha k et al,[13] also had 72.89% males and 27.11% females. The large difference of percentage between males and females can possibly be explained by prevailing social evils like gender discrimination and low female to male sex ratio for various social reasons.

Most of the patients in this study were middle aged adults (41-60 years) i.e. 36%, followed by young adults (20-40 years) who were 34%, 29% were old age (> 60 years) and only 1% were adolescent (< 20 years). The mean age of population in our study was 49.01 years with standard deviation of 17.92 years. The results are similar to the studies done by Reddy et al [17] in which the mean age of study population was 48.8 years and the most common age group being 20 to 60 years of age, another study done in Qatar by Khan et al,[44] it was observed that the mean age group of the study population was 47.4 ± 18.2 years and a study by Arya Shashikant and Archana[45] from India also showed that the mean age of the study population was 38.10 years with most of the study population between 21 and 60 years of age.

48% of patients had duration of present illness of less than 2 weeks, 41% had duration of illness between 14- 42 days (2 weeks to 6 weeks) and the rest 11% had more than 6 weeks history of illness. In a study done by Gupta R et al.[35] also the majority of the study population had 3-4 weeks duration of symptoms before coming to the institution. The most common presenting complaints in this study was shortness of breath (76%), followed by fever (59%), dry cough (53%) and chest pain (49%). Similar results were observed in a study conducted by Reddy et al [17] , the most

common presenting symptom was dyspnea (84%) followed by cough (80%), fever (65%), and chest pain (43%). Another study by Al Alusi[46] included 100 patients in their study, of which the most common symptoms were dyspnea (87%), cough (86%), fever (79%), followed by chest pain (67%). History of trauma was present in 10% of the cases and 4% had hemoptysis. Other complaints like weight loss (10%), loss of appetite (4%), and pain abdomen (1%) were present in 15% of the cases.

Out of the 100 patients, 22% had diabetes mellitus and hypertension each, 10% had a history of tuberculosis in the past, 6% had CLD and 2% had CKD, 4% patients had history of malignancy, coronary artery disease 2% of the cases,1% hypothyroidism, 3% of the patients were HCV reactive, 1% had HBV and another 1% had HIV positive status.1% patient had covid positive history, 1% had history of hysterectomy in the past, 1% patient had history of bronchial asthma and 1% patient had history of ventilatory support in the recent past. In a similar study by Shashikant et al [45] past history of TB was present in 36.4% of cases followed by DM in 27.6% and CLD and CAD in 13.6%.There is high prevalence of diabetes mellitus and hypertension in punjab due to changing lifestyle and other epidemiological factors as observed by RJ Anjana et al[47] and Tripathi et al[48] in their studies.

In this study 27% of the patients had a history of addiction. 17% were addicted to alcohol, 8% were addicted to smoking. 1% had a history of tobacco and another 1% was addicted to opioids. In one study by P Tewatia et al[49] a significant association was seen between smoking duration and tuberculous pleural effusion ($p < 0.001$). Regular alcohol use, history of contact with TB patients, past history of TB, family history of TB and underweight were present in a significantly higher number of cases (tuberculous pleural effusion) than controls ($p < 0.05$ each).

On general physical examination, 43% patients were within normal BMI range (19-22.9 kg/m^2), 29% patients were overweight (between BMI of 23-24.9 kg/m^2), only 1% was underweight (BMI of less than 18kg/m^2). Rest

27% was in the obese category (BMI > 25kg/m^2). In one study by Singh et al[50], it was found that in Punjab population 41.5% subjects were non-obese, 15.9% were overweight, 29.4% were obese and 13.2% were morbidly obese. The prevalence of all the 3 conditions (overweight, obesity and morbid obesity) increased with increasing age in both sexes. Another study done by Girdhar and et al[43] showed the prevalence of overweight and obesity as 12.7% and 29.6% respectively.

On chest examination, the most common findings were asymmetrical chest (81%), tracheal deviation (70%), decreased tactile fremitus (100%), and dullness to percussion (100%). On auscultation, air entry was absent in certain areas on the right side in 51%, at the left side in 42% and bilaterally in 7% according to the amount of pleural fluid. In one study conducted by In Wong CL[5] of the 8 physical examination maneuvers evaluated and included in their studies (conventional percussion, auscultatory percussion, breath sounds, chest expansion, tactile vocal fremitus, vocal resonance, crackles, and pleural friction rub), dullness to conventional percussion was most accurate for diagnosing pleural effusion (positive likelihood ratio, 8.7; 95% confidence interval, 2.2-33.8), while the absence of reduced tactile vocal fremitus made pleural effusion less likely (negative likelihood ratio, 0.21; 95% confidence interval, 0.12-0.37).

Right sided effusion was present in 51%, 42% had left sided effusion and the rest 7% had bilateral effusion. The study conducted by Adeoye et al[51] also had most exudative pleural effusions as right sided (50.2%), followed by left sided effusions (42.7%) and bilateral were 5.6% of the total cases. Another study by Dhital et al [52] and Selvamani et al [53] concluded right sided effusions were most common in their studies. It can be explained by the fact that the right lung is larger and has more pleural surface area therefore more secretion of pleural fluid. Right bronchus is vertical and in line with trachea than the left therefore infectious material tends to enter the right lung more leading to local pathology and pleural effusion. Major

openings like vena-caval hiatus as well as minor openings are more in the right hemi-diaphragm than on the left side.

On the USG chest of the 100 patients 13% patients had loculated pleural effusion (single locule), 17% had multiloculated or septated effusion. It was observed in study conducted by RC Bandaru and et al[54] exudates were complex septated (62.5%), echogenic (25%) or complex non-septated (8.9%) on USG with very few being anechoic (3.5%) and loculations were better appreciated on ultrasound. However Asciak and colleagues[**55**] demonstrated in their own prospective series that the specificity of "echogenicity" in identifying an exudative effusion over a transudate was only 57.1%. In their series of the 140 cases, they identified six (7%) patients with echogenic effusions that were ultimately diagnosed as a transudate.

USG guided diagnostic pleural tap was done. 3% developed hydropneumothorax and 1% developed hemothorax of the total 100 patients in our study.In a study done in ICU patients by Rodrigues.L et al[56] there was one pneumothorax, for a prevalence rate of mechanical complications in this population of 1.2%. In another case series done by RZ Ibrahim et al[57] it showed that the ultrasound guided procedure for tapping pleural effusion were easy to do, make patients convenient, safe and shorten hospitalization after cardiac surgery. Pneumothorax is a known complication of USG guided pleural tap seen in less than 5% of the patients.

All the patients had pleural fluid analysis and all were exudative as per the study criteria. Out of the 100 cases studied, the most common etiology in our study was tuberculosis 42%, then parapneumonic effusion 16%,empyema -16%, empyema -15%, malignant pleural effusions 9%, others 11% (pulmonary embolism, hemothorax, parasitic, viral and chylothorax) and unknown etiology 7%. In a study done by Reddy et al[17] the most common etiology was- tuberculosis (38%), followed by parapneumonic effusion (28.5%) then malignant pleural effusion (22.2%).

Three patients had chylothorax, two patients had pancreatic pleural effusion, and the diagnosis was unknown in two patients. In another study conducted by Rahul et al [35] it was observed that of the total 1000 patients, 69.5% had tuberculosis followed by malignancy (16%). Also in study by Maikap and et al[4] tuberculosis was the most common cause (68.8%), followed by malignancy (14%), empyema (6%). The lower percentage of malignant pleural effusion in our study can possibly be explained by the fact that Dayanand medical college is a referral institute and here we have a separate dedicated oncology center. Most of the patients are on follow up and not everyone undergoes diagnostic pleural tap, therefore a bias in taking cases can be the possible reason.

In tubercular pleural effusion pleural fluid was predominantly straw colored in 83.3% and rest 16.6% were pus. Mohan et al[58] and Raghavan et al[59] in their studies also observed that tuberculous effusions were grossly straw colored in appearance. These were lymphocytic predominant in 95.24% and the rest 4.76% had neutrophil predominance. It was seen that lymphocyte to neutrophil ratio of more than 0.75 had sensitivity of 61.29% and specificity of 89.47% in diagnosing tuberculous pleural effusion. Sensitivity of ADA for diagnosing pleural effusion was 56.76% and specificity of 100% with a positive predictive value of 100% and negative predictive value of 44.83%. Raised ADA however can be increased in other etiologies as well lile rheumatoid pleurisy and empyema. If we combine above two i.e. ADA > 40 U/Land lymphocyte to neutrophil ratio of more than 0.75 the test sensitivity increases to 74.51% with specificity of 91.84% for diagnosing tuberculous pleural effusion with a PPV of 90.48% and NPV of 77.59%. Rajneesh et al[60] also inferred from their study that pleural fluid lymphocyte to neutrophil ratio >0.75 is an efficient means of diagnosing tuberculous pleural effusion and its combination with ADA level gives us more accuracy and surety about the diagnosis of tubercular pleural effusion. In similarly study by Rahman.F et al[61] it was observed that ADA of more than 50 IU/L had sensitivity of 97.6%, specificity 87.5%,

positive predictive value 97.6%, negative predictive value 87.5% which increased to 100% and 92.8%, 98.6% and 100% respectively in combination with test of Lymphocytic/Neutrophilic ratio >0.75. So the use of raised ADA alone in the absence of lymphocyte predominance or other evidence of tuberculosis should be restricted in diagnosing tuberculous pleural effusion. CBNAAT was positive in 11 patients (n-42, 26.1% in its group) with sensitivity of 26.19% and specificity of 100%. PPV was 100% and NPV was 65.17%. In studies done by Birajdar et al[62], Kausal et al[63] and Godwin et al[64] CBNAAT was found to be positive in 2 former and 5 in the latter two studies therefore confirming the presence of tuberculous pleural effusion. Positive pleural fluid AFB observed in 2 patients (4.76% in TB group),1 patient had lymph node tuberculosis on biopsy and another 1 had chronic granulomatous inflammation, sputum AFB was positive in 2 patients (4.76%) and mantoux test was positive in 5 (11.9%). In a study done in Qatar by Zahid et al[65], positive AFB was observed in pleural fluid (13%), and sputum (16%). Chakraborty and et al[34], also had pleural fluid CBNAAT positive for in 24 patients (32%). Sputum smear for acid fast bacilli (AFB) was positive in 3 (4%) patients. Tuberculous pleural effusion is type IV hypersensitivity reaction and mycobacterial load in pleural fluid is usually low. Pleural fluid AFB has sensitivity of 5%[66].Therefore CBNAAT and AFB pleural fluid, owing to its low sensitivity, should not be included in the diagnostic protocol of Tuberculous pleural effusion in high prevalence areas. Total of 9 (21.4% of the TB cases) patients with tuberculous pleural effusion required intervention. 6 (14.2%) patients required only percutaneous drainage while 3 (7.1%) patients required both percutaneous drainage and flushing with fibrinolytic agent- streptokinase. The pleural drainage was done only when patients were having breathing difficulty or if there was indication on imaging or pleural fluid analysis as routine use of catheter drainage and fibrinolytic therapy does not appear to affect long term outcomes as demonstrated by Lai YF et al[67] in a randomized trial having 61 patients with newly diagnosed tuberculous

pleural effusion treated with antituberculous therapy with or without catheter drainage.1 patient in tuberculous pleural effusion group expired while 64.28% had complete resolution at 3 months. 8 patients were put on modified ATT due to adverse effects of the treatment. In one retrospective study by Abbara et al[68], done at a tuberculosis center DILI was seen in 105 (6.9%) cases of amongst 1529 patients diagnosed with active TB between April 2010 and May 2014. Risk factors include low body weight, alcohol ingestion, poor nutrition, genetic factors etc.

16% parapneumonic effusions and 15% empyema were seen in this study. Most of these were neutrophil predominant with pleural fluid LDH to serum LDH ratio more than 1 in 100 % parapneumonic and 88.87 % in empyema. Glucose was less than 40 mg/dl in 66.7% empyema cases and 75% of parapneumonic cases. In a study by Banga et al[69], biochemical examination of pleural fluid revealed the exudative nature of the fluid with low glucose in all patients. A majority of the patients (n = 26, 83.9%) had neutrophil dominance in the pleural fluid. Higher levels of pleural fluid LDH and pleural fluid to serum LDH ratio was also observed in study by Ernam D et al[70]. The biochemical analysis helps in guiding the treatment and requirement of drainage. Pleural fluid cultures were positive in 21% of the cases, most common organism isolated was MRSA i.e. methicillin resistant Staph aureus accounting for the 8% of the total cases studied, followed by E. coli (4%), Klebsiella pneumoniae (3%), Pseudomonas aeruginosa (2%) Acinetobacter (2%), Burkholderia (1%) and polymicrobial growth (1%) (both Streptococcus pneumoniae and Pseudomonas aeruginosa were grown in culture). Banga et[69] also had 13 positive culture reports of which Staphylococcus aureus was the commonest (6 patients, 46.2%). Rest of the patients Pseudomonas aeruginosa (n = 5), Klebsiella pneumoniae (n = 4) and Escherichia coli (n = 2). More than one organism was isolated in four patients. Over the years, the use of antibiotics have given rise to methicillin resistant staph aureus both in hospitals and community as well. 35 patients required drainage and 13

patients required intrapleural streptokinase injection for adequate drainage.It promoted early and better treatment outcome in patients along with the antibiotics. Bouros D et al[71] in their study also concluded that intrapleural instillation of streptokinase is an effective and safe mode of treatment for complicated parapneumonic effusions and pleural empyemas and alleviates the need for thoracotomy. The current indications for intrapleural fibrinolytics include an occluded small-bore catheter, decrease or cessation of pleural fluid drainage in the context of a complex pleural space, a multiloculated pleural space, and as a trial before committing the patient to surgery. Fibrinolytic agents would probably be most effective in the early fibrinolytic stage in avoiding the need for surgical drainage.[72] The overall mortality in these group at 3 month period was 21.8% which is comparable to the studies done by Arivudainambi VP et al[73] in which the observed mortality was 30.50% and and another study by Kookoolis AS et al[74] in which at 30-days, 15% of the patients had died and by 12-months mortality had increased to 32%.The mortality increases in these patients as parapneumonic effusions and empyema correlates with more advanced stages of pneumoniae.

9 patients of the 100 were found to have malignant pleural effusion in this study. Pleural fluid appearance was hemorrhagic in 33.33% of the cases. It was exudative (100%) lymphocytic predominant in 88.88% of the cases with lymphocyte to neutrophil ratio of more than 0.75 in 77.77% cases. Porcel[75] in his study also demonstrated that bloody pleural effusion was present in his study in only 11% of the patients. When pleural injury becomes long-standing, the fluid is generally lymphocytic predominant (>50% of the leukocytes) as seen in malignancy or TB. ADA value of greater than 40 U/L was found in 55.6% cases with a p value of 0.186 which is statistically non-significant. Also, the combined value of lymphocyte to neutrophil ratio of > 0.75 and ADA > 40 U/L was found in only 10.2% of the cases against 75.5% as seen in tuberculous pleural effusion. Pleural fluid cytology for malignant cells was positive in 77.77%

cases with sensitivity of 77.78% and specificity of 100% with PPV of 100% and NPV of 97.85%. Nonspecific inflammatory effusion secondary to subpleural intraparenchymal lung tumor development, lymphatic vessel obstruction, or immune-mediated inflammation can explain the failure of a cytology examination to provide a diagnosis in some cases. In the latter scenario, the availability of rapid and reliable proof of malignancy by less-invasive procedures is a constant goal, especially in the case of patients who have a prior history of cancer and who develop pleural effusion during follow up[76]. In a study by Kaul V et al[57] , pleural fluid cytology had sensitivity of 46-59% and specificity of 100%. Irani et al[78], also demonstrated positive cytology results in 96% of the cases and adenocarcinoma being the most common malignancy. In this study also the most common cause was found to be adenocarcinoma lung which is consistent with another study done by Mandal S et al,[79] Even after significant progress in therapeutic approach, the long term prognosis are severe with mean survival of 4-9 months from diagnosis of malignant pleural effusions as seen in study by Merrigi et al,[80] The presence of malignant tumor cells in the pleural fluid is generally indicative of advanced disease and is associated with high morbidity and mortality with reduced therapeutic options[80].In this study also total 33.33% expired at 3 month follow up after initial diagnosis of malignant pleural effusions.

11% patients were categorized under the heading of 'other' causes. Of these 3% were due to pulmonary embolism and had lymphocytic predominant exudative pleural effusion. 2 were hemorrhagic and 1 had straw colored appearance. CT chest was the diagnostic modality of choice for pulmonary embolism. CT chest was done in 27 patients in this study and pulmonary embolism with pleural effusion was present in 11.11% of these. All patients were managed with low molecular weight heparin. In a study by Jackson K et al[81] all the effusions due to pulmonary embolism were bloody. Of the total CTPA done 19.4% had pulmonary embolism and 5.4% had both pulmonary embolism and pleural effusion. In study by

Porcel et al [82] found that pleural effusions associated with pulmonary embolism are always exudates. It was also observed by Candeira et al[83] that fluid characteristics, were non-specific and unhelpful in making the diagnosis and given a high index of clinical suspicion or in the context of an effusion that remains undiagnosed after standard baseline investigations, possibility of pulmonary embolism should be pursued radiologically. In another study by Liu M et al[84] the incidence of pleural effusion was significantly higher in patients with pulmonary embolism (243/1220, 19.9%).

Hemothorax was diagnosed in 3% of the cases. History of trauma was present in all the 3 cases. Mean LDH of pleural fluid was 640 U/L. Intercostal drain insertion was done in all. 1 patient expired in hospital while the other 2 (66.67%) had VATS. Both had resolution at 1 month follow up and developed pleural thickening as seen on follow up chest x ray. Bhattacharyya DK et al[85], also observed in their study that all patients had a history of trauma (in some form). 80.51% required drainage via large bore tube thoracostomy and 9.32% patients required thoracotomy. On follow up all complaints usually disappear within one month after discharge and follow- up chest X-ray did not show any residual collection. Hemorrhagic pleural effusion is one of the common causes of pleural thickening.

2 patients in our study having right sided effusion with anchovy sauce appearance were due to Amoebiasis. Both had positive serology tests for Entamoeba Histolytica and subsequently improved on clinical follow up when given antibiotics and antimalarial agents. Intercostal drainage was done in both of these. In a study by Meng XY et al[86] , also had 79 cases of amoebic liver disease with pleuropulmonary involvement. Metronidazole combined with needle aspiration and/or percutaneous catheter drainage is the treatment of choice for amebic liver abscess with perforation. Karkhanis VS[10] et al in their study also observed that usually parasitic

effusion was right-sided and the fluid had anchovy sauce appearance and is exudative with polymorphonuclear predominance.

2 patients in this study were attributed to have pleural effusion due to SARS cov 2 virus. Both had bilateral pleural effusion and covid RTPCR was positive in both of them. Mean pleural fluid LDH was 845.5 U/L. CT chest were suggestive of extensive ground glass opacities in both of them.1 of these patients expired at follow up. Woon H et al[87] also described COVID-19-related pleural effusions as exudative, lymphocytic or neutrophil predominant pleural fluid with markedly elevated LDH levels and pleural fluid to serum (PF:S) LDH ratio. PF:S LDH ratio of 1.3 and more to be suggestive of COVID-19-related pleural effusions. Sammun et al[88], also noted exudative effusion of either lymphocytic or neutrophil predominant in their case series. All the effusions were sanguineous on appearance and on biochemistry pleural fluid LDH was markedly elevated compared to serum LDH among several COVID-19 patients that received pleural drainage. Zhan et al[89], also concluded in their study that COVID patients with pleural effusion might have severe inflammation and a poor prognosis. SS rathore et al[90] in their study on COVID 19 related pleural effusions concluded that the presence of pleural effusion can assist as a prognostic factor to evaluate the risk of worse outcomes in COVID-19 patients hence, it is recommended that hospitalized COVID-19 patients with pleural effusion should be managed on an early basis.

One patient was diagnosed to have chylothorax. Patient was diagnosed case of Hodgkin's lymphoma and was on treatment. He developed shortness of breath. On evaluation he had bilateral chylous effusion with triglycerides level of 240 mg/dl. Intercostal drainage was put in. Chemotherapy subsequently continued. Verma SK et al[91] reported a case of chylothorax as a presenting complaint in a patient with Hodgkin's lymphoma.Nikbakhsh et al[92] also reported dyspnea to be the most common symptom present in patients with chylothorax. All the patients in their study had triglycerides more than 110 mg/dl.

7% patients even after evaluation remained undiagnosed. In one retrospective study by Venekamp et[93] al on 75 patients with unknown etiology who were followed for 2 years and majority (91.7%) had benign course and spontaneous resolution of the effusion in 81.8% of cases. In our study also one patient of these was diagnosed with malignancy (lymphangitis carcinomatosis) at 1 month period of follow up and was started on chemotherapy. 3 patients had resolution at 3 months, while the rest had recurrent or persistent effusion. During follow up one patient required percutaneous drainage while 2 required VATS.

Analysis of data between survivor and non survivor groups suggested that empyema was the leading cause of death followed by malignancy and parapneumonic effusion. Older age (57.62 years with standard deviation of 12.67 years), female gender were the risk factors for mortality .This can be explained by difference in the immunity between young and old patients and presence of comorbidities among the older population might be a confounding factor. Among the chief complaints , shortness of breath, fever , cough and expectoration were the most common complaints, however complaint of expectoration was statistically significant with increased mortality perhaps because of the involvement of primary lung parenchyma. Moreover empyema and parapneumonic effusions are the advanced stages of underlying pneumoniae. Patients with multisystem involvement had greater risk of mortality like presence of jaundice and encephalopathy at hospitalization were statistically significant in view of poor outcome. Other predictors of higher mortality which were significant in this study were higher TLC, high creatinine more than 1.1 mg/dl, decreased albumin (< 3.5 mg/dl) and raised serum (LDH >200 u/l). On pleural fluid analysis low glucose that is less than 40g/dl was statistically significant in the non survivor group. Pleural fluid LDH, TLC and protein were not significant in differentiating survivor and the non survivor group. Presence of multiloculations or septations was another risk factor for higher mortality. These results were comparable to a study done by Tsai

YM et al[94], Ozyurtkan MO et al[95] also observed that high-risk tumors, a poor performance status, and lower pleural fluid glucose concentration are predictors of the early mortality within three months in the patients with a malignant pleural effusion.

SUMMARY AND CONCLUSION

Clinical profiles of 100 patients admitted with exudative pleural effusion as per the inclusion criteria were prospectively observed and followed.

1. Maximum number of patients (36%) were in the age group of 40 to 60 years.
2. Males were found to be 73% and 27% were females.
3. 49% patients had duration of stay in hospital between 10-30 days
4. The most common symptom at presentation was shortness of breath (76%), fever (59%), cough (53%), chest pain (49%), expectoration (18%), trauma (10%) hemoptysis (4%).
5. Right sided effusions were present in 51%, 42% had left sided effusions and the rest 7% had bilateral effusion.
6. Most of the pleural fluids were straw coloured followed by pus then hemorrhagic, two had anchovy sauce appearance and one had chylous appearance.
7. Tuberculous pleural effusion was the most common cause (42%) followed by parapneumonic (16%), empyema (15%), malignant (9%), other causes (11%) and unknown in 7% patients.
8. In diagnosing tuberculous effusion the combined sensitivity of pleural fluid LDH to serum LDH and ADA was 74.51% and specificity was 91.84%. It is therefore an efficient means of diagnosing tuberculous pleural effusion. Use of ADA alone should be avoided in diagnosing tuberculous pleural effusion.
9. In parapneumonic effusion and empyema, these were mainly neutrophil predominant with the majority having glucose less than 40 mg/dl.
10. The most common organisms grown in pleural fluid cultures were MRSA i.e. methicillin resistant Staph aureus, followed by E. coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Acinetobacter, Burkholderia.

11. Majority of the malignant pleural effusions were lymphocytic predominant. The combined value of lymphocyte to neutrophil ratio of > 0.75 and ADA > 40 U/L was found in only 10.2% of the cases against 75.5% as seen in tuberculous pleural effusion.
12. Pleural fluid cytology for malignant cells was positive in 77.77% cases with sensitivity of 77.78% and specificity of 100% with PPV of 100% and NPV of 97.85%. Adenocarcinoma lung was the most common etiology in malignant pleural effusion.
13. For diagnosing pleural effusion due to pulmonary embolism, pleural fluid analysis is not significant and CTPA is the investigation of choice.
14. Hemothorax is a common indication for VATS.
15. Serological tests like entamoeba serology along with the clinical context that is right sided exudative effusion with anchovy sauce appearance helps in diagnosing parasitic effusion due to parasitic etiology.
16. Pleural effusions due to covid 19 are exudative with high LDH and are predictor of poor mortality. More studies and research work is required for characterizing pleural effusion due to covid 19 and its outcome in patients.
17. Chylothorax is one of rare causes of exudative pleural effusion, mostly related to trauma. Amongst the non traumatic causes, lymphoma is the leading cause.
18. Despite intensive evaluation 7% patients remained undiagnosed exudative pleural effusions. One was later re-classified as malignant pleural effusion.
19. Most of the pleural effusions resolve with treatment. Drainage and fibrinolytic therapy and sometimes VATS are required as per the clinical indication. treatment is palliative in case of malignant effusions.

20. Overall mortality was 13%, empyema, followed by malignant pleural effusion and parapneumonic effusions were the leading causes.

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PROFORMA

NAME-	AGE/SEX-
DATE OF ADMISSION-	DATE OF DISCHARGE-
MRD NO-	OCCUPATION-
ADDRESS-	SOCIO ECONOMIC STATUS-

HISTORY	SYMPTOM	Y/N	DUR	SYMPTOM	Y/N	DUR
PRESENT HISTORY	CHEST PAIN			FEVER		
	SOB			CHEST TRAUMA		
	COUGH			HEMOPTYSIS		
	EXPECTORATION			OTHER		
PAST HISTORY	TB			CLD/ALD		
	HTN			CKD		
	DM			OTHER		
EXPOSURE TO DRUGS/TREATMENT	NIROFURANTOIN			MTX		
	AMIODARONE			OHT		
	PHENYTOIN			RADIOTHERAPY		
	OTHER					
PERSONAL HISTORY	SMOKING			MENSTRUAL HIS		
	ALCOHOL			STD EXPOSURE		
	OTHER					

GENERAL PHYSICAL EXAMINATION ON ADMISSION

APPEARANCE-	SENSORIUM-	HT-(m)	WT-(kg)-	BMI-(kg/m^2)
TEMPERATURE-	PAIN-	HR	RR	BP-
PALLOR-	ICTERUS-	CYANOSIS-	LAP-	
CLUBBING-	JVP-	EDEMA-		

SYSTEMIC EXAMINATION ON ADMISSION-

CHEST	SYMMETRY-	TRACHEA-	APICAL IMPULSE-
	TACTILE FREMITUS-	PERCUSSION-	PLEURAL RUB-
	AUSCULATION-		

CVS	
CNS	
PER ABDOMEN-	
ANY LOCAL FINDING	

INVESTIGATIONS

HB	PCV-	TLC-	DLC-	PLATELETS
CREATININE-	NA/K -	S. ALBUMIN-	S.PROTEIN	PTI INR
UREA-	RBS-	BILLIRUBIN(T/D)-	ALT/AST-	ALP-
ESR	MONTOUX TEST	URINE:PROTEIN GLUCOSE DEPOSITS-	S. LDH-	TSH/T3/T4-

RADIOLOGICAL INVESTIGATIONS/FINDINGS& INTERVENTIONS

CHEST X RAY				
USG CHEST	LATERALITY-			
	QUANTITY-	MILD	MODERATE	SIGNIFICANT
	SEPTATIONS-			
	LOCULATION-			
CECT/HRCT CHEST				
US GUIDED THORACOCENTESIS-	DIAGNOSTIC / THERAPEUTIC AMOUNT OF FLUID ASPIRATED -			

INTERVENTIONS

US GUIDED THORACOCENTESIS-	DIAGNOSTIC/THERAPEUTIC-
	AMOUNT OF FLUID APIRATED-
POST PROCEDURE CHEST XRAY/COMPLICATIONS -	

PLEURAL FLUID ANALYSIS-

MACROSCOPIC-	COLOR- HEMORRHAGIC/STRAW/PUS
	OTHER-

SAMPLE	PROTEIN	GLUCOSE	LDH	PH	OTHER
P.FLUID					
SERUM					

TLC-	DLC-	RBC-	PCV-	AFB-
PCR-	C/S-	M CELL-	ADA-	MISC-

ANALYSIS-

OTHER INVESTIGATIONS(if indicated)

ANA(IF)-	RA FACTOR-	ANTI CCP
ANA (PROFILE)-	C3/C4 LEVELS-	MISC

OTHER INTERVENTIONS /PROCEDURE–

FINAL DIAGNOSIS-

OUTCOME

TREATMENT	DATE OF INITIATION	DURATION OF SYMPTOMS	DELAY IN INITIATION FROM 1ST SYMPTOM-
ANTIBIOTICS			
ATT- CATEGORY			
CHEMOTHERAPY/RADIOTHERAPY			
SURGICAL MANAGEMENT			
OTHER			

FOLLOW UP

	AT DISCHARGE	1 MONTH	3 MONTH
RESOLUTION OF SYMPTOMS			
CXR PA VIEW			
ADVERSE OUTCOME/COMPLICATION/ OTHER			

S.NO	NAME	MRD	AGE	GENDER	S/E STATUS	OCCUPATION	DURATION OF HOSPITALISATION	PRESENT HISTORY								DURATION	PAST HSITORY								PERSONAL HISTORY			OTHER	BMI
							DAYS	CHEST PAIN	SOB	FEVER	TRAUMA	COUGH	EXPECTORATION	HEMPOTYSIS	OTHER	DAYS	TB	HTN	DM	CLD	CKD	PLHIV	HCV	HBV	OTHER	SMOKE	ALCOHOL	MENSTRUAL HISTORY		KG/M2
1	TW	912927	55	F	UMC	D	12	N	Y	N	N	Y	N	N	N	150	N	Y	N	N	N	N	N	N	CA LUNG	N	N	PM	N	18
2	HK	909231	56	M	UMC	D	7	Y	Y	Y	N	N	N	Y	N	90	N	Y	N	N	N	N	N	N	N	N	N	NA	N	22
3	RK	912477	56	M	UMC	B	13	N	Y	Y	N	Y	Y	N	N	25	N	Y	Y	N	N	N	N	N	N	N	Y	NA	N	26
4	JS	918086	65	M	LMC	D	7	N	Y	Y	N	N	N	N	WT LOSS	15	N	N	N	N	N	N	N	N	CAD	N	Y	NA	N	23.7
5	KL	917448	37	M	LMC	DRIVER	15	Y	Y	Y	N	Y	N	N	N	15	N	N	N	N	N	N	N	N	N	N	N	NA	OPIOID	24
6	JS	731444	44	M	LMC	B	20	N	Y	N	N	Y	N	N	N	15	N	N	N	Y	N	N	N	N	N	N	Y	NA	N	25.4
7	GS	893194	21	M	UMC	D	7	N	Y	Y	N	Y	N	N	N	30	N	N	N	N	N	N	N	N	N	N	N	NA	N	20.2
8	VK	924210	26	M	UMC	D	10	Y	Y	Y	N	Y	Y	N	N	120	N	N	N	N	N	N	N	N	N	N	N	NA	N	21.8
9	CS	810852	68	M	LMC	D	5	N	Y	Y	N	Y	N	N	N	49	N	Y	Y	N	Y	N	N	N	N	N	N	NA	N	25.4
10	DK	426738	52	F	UMC	D	33	N	Y	Y	N	N	N	N	N	3	N	Y	N	N	Y	N	N	N	N	N	N	PM	N	26.2
11	JS	266276	29	M	UMC	B	10	N	Y	Y	N	Y	N	N	WT LOSS	90	N	N	N	N	N	Y	Y	Y	N	N	Y	NA	N	19.8
12	PS	890027	50	M	UMC	B	9	Y	Y	Y	N	Y	N	N	AP LOSS	365	N	N	N	N	N	N	N	N	N	N	N	NA	N	24.1
13	RS	1128967	24	M	UMC	D	35	Y	Y	N	Y	N	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	NA	N	23.1
14	MS	821794	22	M	UMC	ATHLETE	5	Y	N	Y	Y	N	N	N	N	20	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.1
15	SS	796639	54	M	UMC	B	8	N	Y	N	N	Y	N	N	WT LOSS	30	N	Y	N	Y	N	N	N	N	N	N	Y	NA	TOBACOO	26.2
16	S	928716	32	M	UMC	B	6	Y	N	Y	N	N	N	N	WT LOSS	15	Y	N	N	N	N	N	N	N	N	N	N	NA	N	29
17	IS	928766	55	M	LMC	FA	7	N	Y	N	N	N	N	N	AP LOSS	5	N	N	Y	N	N	N	N	N	N	N	Y	NA	N	27
18	AS	928486	51	M	LMC	FA	22	Y	Y	Y	N	Y	Y	N	N	15	N	N	Y	N	N	N	N	N	N	N	N	NA	N	26
19	G	928806	36	F	ULC	DWW	20	N	Y	Y	N	Y	Y	N	N	7	Y	Y	N	N	N	N	N	N	COVID POSITIVE	N	N	R	N	22
20	S	923230	23	F	LMC	D	55	N	Y	Y	N	Y	Y	N	N	5	N	N	N	N	N	N	N	N	ON VENTILATORY	N	N	R	N	19
21	N	921731	51	F	UMC	D	3	Y	N	Y	N	Y	N	N	N	7	N	N	N	N	N	N	N	N	N	N	N	PM	N	23.4
22	HS	921564	84	M	UMC	D	5	Y	N	Y	N	N	N	N	N	15	Y	N	Y	N	N	N	N	N	N	N	N	NA	N	24.9
23	TD	812649	73	F	UMC	D	5	N	N	N	N	Y	N	N	N	7	N	N	Y	N	N	N	N	N	CA OVARY	N	N	PM	N	20.7
24	HM	779489	62	M	LMC	D	7	N	Y	Y	N	Y	N	N	N	12	Y	N	N	N	N	N	N	N	N	Y	N	NA	N	24
25	PK	294121	58	F	UMC	D	13	N	Y	N	N	N	N	N	AP LOSS	20	N	N	Y	N	N	N	N	N	N	N	N	PM	N	23.9
26	KS	932735	67	M	LMC	D	4	Y	Y	N	N	N	N	N	N	10	N	Y	N	N	N	N	N	N	N	N	N	NA	N	27.14
27	GS	934685	18	M	ULC	D	17	Y	N	Y	N	N	N	N	N	15	N	N	N	N	N	N	N	N	N	N	N	NA	N	27.1
28	K	935693	24	F	UMC	D	9	N	Y	Y	N	Y	N	N	N	60	N	N	N	N	N	N	N	N	N	N	N	R	N	21.6
29	D	950085	36	F	UPPER	D	8	N	Y	N	N	Y	N	N	N	10	N	N	Y	N	N	N	N	N	HYPOTHYROIDISM	N	N	R	N	23
30	SKA	592481	62	M	UMC	D	12	N	Y	N	N	N	N	N	N	24	N	Y	Y	N	N	N	N	N	N	N	N	NA	N	27.8
31	JS	731444	45	M	LMC	B	20	Y	Y	N	N	N	N	N	N	15	N	N	N	Y	N	N	N	N	N	N	Y	NA	N	20.8
32	GS	941024	60	M	UMC	B	22	Y	N	Y	N	N	N	N	N	80	N	N	N	N	N	N	N	N	N	N	N	NA	N	27.2
33	AS	943064	73	M	UMC	D	13	Y	N	N	Y	Y	N	N	WT LOSS	15	N	N	N	N	N	N	N	N	N	N	Y	NA	N	23.8
34	BK	815977	40	F	LMC	D	17	N	N	Y	N	Y	N	N	N	20	N	N	N	N	N	N	N	N	N	N	N	R	N	22.9
35	DK	939697	35	M	UMC	B	15	Y	Y	N	N	Y	N	N	N	90	N	N	N	N	N	N	N	N	N	Y	N	NA	N	18.1
36	SS	931901	36	M	LMC	DRIVER	5	Y	N	Y	N	N	N	N	N	5	N	N	N	N	N	N	N	N	N	N	N	NA	N	24
37	S	928716	32	M	UMC	B	18	Y	Y	Y	N	Y	N	N	N	30	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.7
38	KS	779489	67	M	UMC	D	13	Y	Y	N	N	N	N	N	N	10	N	Y	N	N	N	N	N	N	N	N	N	NA	N	25.8
39	MK	924818	26	F	UMC	D	2	N	Y	Y	N	N	N	N	N	25	N	N	N	N	N	N	N	N	N	N	N	R	N	19.8
40	MS	945391	23	M	UMC	ATHLETE	3	Y	Y	N	Y	Y	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	NA	N	20.2
41	UD	949251	56	F	UMC	D	15	N	Y	Y	N	Y	Y	N	N	5	N	N	N	Y	N	N	N	N	N	N	N	PM	N	28.9
42	SK	947895	56	M	UMC	FA	20	N	Y	N	N	N	N	N	N	7	N	N	N	N	N	N	N	N	N	N	N	NA	N	24.9
43	GK	940300	69	F	LMC	FA	1	N	Y	Y	N	Y	Y	N	N	30	N	N	N	N	N	N	N	N	N	N	N	PM	N	18.2
44	KJ	916469	95	F	UMC	D	14	N	Y	N	N	N	N	N	N	10	N	Y	N	N	N	N	N	N	N	N	N	PM	N	21

SPO2 LESS THAN 94 RECQUIRING OXYEGEN	ICTERUS	CLUBBING	LAP	CHEST					CVS	CNS	ABD	OTHER	HB	TLC	DLC	CREAT	ALBUMIN	PROTEIN	OT	PT	ALP	ESR	S .LDH	MANTOUX	CXR				
				SYMMETRY	TRACHEA	TACTILE FREMITUS	PERCUSSION	AUSCULTATION																	EFFUSION	LATERALITY	OPACITY	M.SHIFT	OTHERS
													G/DL	10^3/UL	NE/L/E	MG/DL	G/DL	G/DL	U/L	U/L	U/L		U/L	MM					
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.2	14.3	L	0.5	2.58	5.6	45	46	473	90	374	NEG	Y	RT	N	Y	N
Y	N	N	N	AS	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	11.2	10.8	NE	0.75	2.6	5.2	13	16	124	55	550	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.7	9.5	L	0.9	3.43	6.6	35	39	88	73	178	ND	Y	LT	N	Y	N
N	N	N	N	AS	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.9	14.1	NE	0.89	3.5	7.1	33	43	87	54	187	NEG	Y	RT	Y	Y	N
N	N	N	N	AS	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.8	18.6	E	0.59	2.04	5.1	34	27	185	59	232	ND	Y	RT	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9	10.4	N	1.59	2.37	8	56	193	157	83	268	ND	Y	RT	N	Y	N
N	N	N	N	AS	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	13.3	6.3	L	0.9	4.6	5.2	20	22	45	79	296	ND	Y	RT	N	Y	N
N	N	N	N	AS	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	13.6	7.4	NE	0.9	3.4	4.8	30	31	55	14	200	NEG	Y	LT	N	Y	N
N	Y	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.4	5.3	NE	1.11	3.23	6.2	43	54	88	89	231	NEG	Y	LT	N	Y	N
N	N	N	N	AS	C	DECREASED	DULL	LT ABSENT	NAD	ENCEPH	NAD	NAD	9.3	16.4	NE	14.05	3..21	5.1	33	35	298	11	273	NEG	Y	LT	N	Y	N
N	N	N	Y	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	9	3.7	NE	0.8	2.97	8	134	124	56	74	712	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.1	13	NE	0.6	3.55	7.1	88	40	93	73	262	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.9	7.7	NE	0.56	2.07	5.7	13	34	66	46	163	NEG	Y	RT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	11.1	5.5	NE	0.88	3.8	7.2	24	26	87	52	254	NEG	Y	RT	N	N	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	ASCITIS	NAD	10.1	8.8	NE	1.12	2.4	6.2	48	45	89	62	202	NEG	Y	LT	N	Y	N
N	N	N	Y	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.9	11.8	L	1.11	3.9	7.3	34	38	88	120	192	NEG	Y	RT	Y	N	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.1	5.5	L	0.49	2.5	5.2	78	87	139	106	210	NEG	Y	LT	N	Y	N
Y	N	N	N	AS	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	13.2	19.9	NE	1.14	2.79	7.1	106	77	242	75	254	NEG	Y	RT	Y	Y	PNEUMOTH
Y	N	N	N	AS	S	DECREASED	DULL	B/L ABSENT	NAD	NAD	ASCITIS	NAD	11.3	5.8	NE	0.51	3.8	6.8	87	49	375	54	552	NEG	Y	B/L	Y	Y	GGO
Y	N	Y	N	S	S	DECREASED	DULL	RT ABSENT	NAD	ENCEPH	ASCITIS	NAD	10.1	22	NE	1.1	2.24	5.6	88	98	159	45	154	NEG	Y	RT	Y	N	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.3	6.8	L	0.88	4.1	7.8	46	49	88	49	226	ND	Y	RT	N	N	N
N	N	N	N	S	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.1	7.8	L	1.2	3.2	7.1	48	56	98	89	204	POS	Y	LT	N	N	FIBROSIS
Y	N	N	N	AS	C	DECREASED	DULL	B/L ABSENT	NAD	NAD	ASCITIS	NAD	11.2	8.7	NE	0.99	3.67	6.8	35	45	82	76	145	ND	Y	B/L	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.1	11	L	0.19	2.86	4.91	26	29	153	88	263	POS	Y	LT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.1	6.1	L	0.98	3.63	6.3	55	56	76	14	183	NEG	Y	RT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.6	13.2	NE	0.8	3.53	6.9	38	45	67	115	175	NEG	Y	RT	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.8	23.8	NE	1.11	3.98	7.28	54	67	102	109	281	ND	Y	LT	N	Y	N
N	N	N	N	AS	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.4	5.4	L	0.4	4.43	4.1	193	45	127	9	102	ND	Y	LT	N	N	N
N	N	N	N	AS	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.1	8	L	2.3	3.8	6.4	98	96	88	23	248	ND	Y	RT	N	Y	CAVITY
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.5	7.7	NE	0.05	2.98	4.4	88	54	112	90	180	ND	Y	RT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	ASCITIS	NAD	7.7	4.1	L	2.02	2.8	6.4	38	15	149	67	145	ND	Y	RT	N	Y	N
N	N	N	N	S	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.2	25	NE	1.8	3.7	7.1	56	35	125	98	198	ND	Y	LT	N	N	N
N	N	N	N	AS	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.4	7.8	E	1.01	4.1	7.9	45	35	67	33	143	ND	Y	LT	N	Y	RIB FRACT
N	N	N	N	S	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.1	8.9	NE	0.81	3.7	4	43	34	78	67	134	ND	Y	LT	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.1	7.7	L	0.99	3.2	6.5	57	45	89	98	189	ND	Y	RT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	14.9	9	L	1.04	4.23	7.4	24	25	106	52	177	POS	Y	RT	N	Y	N
N	N	N	Y	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	11.9	7.8	L	0.71	2.88	7.3	35	55	92	120	330	NEG	Y	RT	Y	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	15.1	17	NE	1.1	4.5	8	23	25	67	78	267	NEG	Y	RT	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.1	22	NE	1.12	3.66	7.4	56	58	99	60	199	NEG	Y	LT	Y	N	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	13.3	10.1	NE	0.68	4.8	7	24	23	45	45	180	NEG	Y	LT	Y	N	N
Y	Y	N	N	AS	S	DECREASED	DULL	B/L ABSENT	NAD	ENCEPH	ASCITIS	NAD	12.1	28	NE	3.2	2.8	6.1	67	87	199	89	250	NEG	Y	B/L	Y	Y	CAVITY
Y	N	Y	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.2	12.1	NE	0.99	3.56	6.2	34	23	120	87	193	NEG	Y	LT	Y	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	MUFEL	ENCEPH	NAD	NAD	15.4	16.8	NE	2.11	2.88	5.6	26	29	87	102	405	NEG	Y	RT	Y	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	11.2	12.1	L	0.99	3.12	6.5	12	14	56	100	155	NEG	Y	RT	N	Y	N

USG CHEST						CT CHEST	USG P.TAP		COMPLICATIONS			PLEURAL FLUID ANALYSIS													
LATERALITY	MILD	MOD	SIGNIFICANT	MULTIPLE LOCULATI	ON-SINGLE		DIAGNOSTIC TAP	THERAPEUTIC TAP	Y/N	HEMOTHORAX	H.PNEUMOTHORAX	HEMORRHAGIC	STRAW	PUS	OTHER	CHYLOUS	PROTEIN	GLUCOSE	LDH	PH	TLC	L	NE	L:NE	RBC
																	G/DL	MG/DL	U/L		CELLS/MM3	%	%		CELLS/MM3
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	Y	N	N	N	N	3.6	130	359	ND	300	65	35	1.85	40000
RT	N	Y	N	N	Y	MOD EFFUSION	Y	N	N	N	N	N	Y	N	N	N	4.7	99	1108	ND	200	20	80	0.25	20000
LT	N	Y	N	N	N	ND	Y	N	Y	N	Y	N	N	Y	N	N	5.5	176	360	7	3000	90	10	9	20000
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	2.9	36	900	ND	340	45	55	0.8	1000
RT	Y	N	N	N	Y	ND	Y	N	Y	N	Y	N	N	N	ANCHOVY.S	N	2.7	29	1299	ND	800	30	70	0.42	300
RT	N	N	Y	N	N	SIGNIFICANT RT P.E	Y	Y	N	N	N	N	Y	N	N	N	4.7	73	301	7.5	900	90	10	9	300
RT	N	Y	N	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	3	91	359	ND	400	90	10	9	100
LT	N	Y	N	N	N	ND	Y	N	N	N	N	N	N	Y	N	N	2.8	66	600	ND	780	30	70	0.42	100
LT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.3	77	300	ND	100	70	30	2.3	10
LT	N	Y	N	Y	N	ND	Y	Y	N	N	N	N	N	Y	N	N	3.2	72	273	7.2	300	10	90	0.1	100
LT	N	Y	N	N	N	MOD LT P.E + NEC LAP	Y	N	N	N	N	N	Y	N	N	N	4.9	81	541	ND	700	70	30	2.3	200
LT	N	Y	N	N	N	SIGNIFICANT LT P.E	Y	Y	N	N	N	N	Y	N	N	N	5.4	83	738	7.3	500	80	20	4	800
RT	N	N	Y	N	N	SIGNIFICANT RT P.E	Y	N	N	N	N	Y	N	N	N	N	3.9	88	990	7.2	100	25	75	0.3	100000
RT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	5.5	102	509	7.5	500	90	10	9	1000
LT	N	Y	N	N	Y	MOD LT EMPYEMA	Y	N	N	N	N	N	N	Y	N	N	4.8	42	900	7.2	500	80	20	4	100
RT	Y	N	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	6.2	2	4705	ND	800	5	95	0.5	0
LT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	2.9	136	170	ND	250	70	30	2.3	100
RT	N	Y	N	N	N	RT H.PNEUMOTHORAX WITH CONSOLIDATION	Y	N	N	N	N	N	Y	N	N	N	4.8	10	1900	ND	1200	10	90	0.1	100
B/L	N	Y	N	N	N	EXTENSIVE GGO - CORAD 6, B/L MOD P.E	Y	Y	N	N	N	N	Y	N	N	N	3.5	66	802	ND	400	80	20	4	40000
RT	Y	N	N	Y	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.4	22	1384	7	3000	30	70	0.42	10000
RT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	6.5	293	226	ND	200	90	10	9	100000
LT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.4	198	201	ND	500	70	30	2.3	80
B/L	N	Y	N	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	5.2	83	370	ND	2500	10	90	0.1	10000
LT	N	Y	N	N	N	ND	Y	N	Y	N	Y	N	Y	N	N	N	3.5	65	200	ND	400	70	30	2.3	100
RT	N	N	Y	N	N	SIGNIFICANT LT P.E	Y	N	N	N	N	N	Y	N	N	N	4.5	101	184	ND	600	90	10	9	0
RT	Y	N	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	5.4	18	1590	ND	2000	20	80	0.25	2000
LT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	5	2	1809	ND	5000	10	90	0.1	200
LT	Y	N	N	N	Y	[illegible] P.E RT	Y	N	N	N	N	N	N	Y	N	N	2.9	77	198	ND	200	95	5	19	100
RT	N	Y	N	N	N	MOD RT P.E WITH CAVITATORY LESION	Y	N	N	N	N	N	Y	N	N	N	4.8	88	198	ND	200	60	40	1.5	2000
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	2.3	77	228	ND	500	90	10	9	100
RT	N	N	Y	N	N	SIGNFCNT RT P.E	Y	N	N	N	N	N	Y	N	N	N	4.7	73	301	7.5	700	65	35	1.85	80
LT	Y	N	N	Y	N	ND	Y	N	Y	Y	N	N	N	Y	N	N	5.1	61	502	ND	900	20	80	0.25	500
LT	N	Y	N	N	N	ND	Y	N	N	N	N	Y	N	N	N	N	6.3	90	490	ND	800	90	10	9	50000
LT	Y	N	N	N	N	MILD LT P.E	Y	N	N	N	N	N	Y	N	N	N	2.7	88	230	ND	200	45	55	0.81	90
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	4.5	98	324	ND	500	60	40	1.5	1000
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	5.6	107	969	ND	800	90	10	9	700
RT	N	N	Y	N	N	ND	Y	N	N	N	N	N	N	Y	N	N	6.2	2	3700	7	800	5	95	0.5	0
RT	Y	N	N	N	Y	ND	Y	N	N	N	N	N	N	Y	N	N	5.5	23	1946	ND	1600	10	90	0.1	300
LT	N	Y	N	N	Y	ND	Y	N	N	N	N	N	Y	N	N	N	5.4	20	1100	ND	2500	25	75	0.3	10000
LT	N	Y	N	N	N	MOD LT P.E+ PULM EMBOLISM	Y	N	N	N	N	Y	N	N	N	N	3.8	86	201	ND	200	80	20	4	18000
B/L	N	N	Y	Y	N	ND	Y	Y	N	N	N	N	N	Y	N	N	4.4	29	1504	ND	900	55	45	1.2	880
LT	N	Y	N	N	N	CA LUNG WITH MOD LT P.E	Y	Y	N	N	N	N	Y	N	N	N	3.5	89	345	ND	100	90	10	9	1000
RT	N	Y	N	N	Y	ND	Y	N	N	N	N	N	N	Y	N	N	5.1	2	885	ND	1600	30	70	0.4	10000
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	4.1	80	345	ND	600	90	10	9	100

												DX	TB				M. P.E		EMPYEMA		PP.E				CHYLOTHORAX
PCV	AFB	GRAM STAIN	CBNAAT	C/S	M CELL	ADA	TG	OTHER	HISTOPATHOLOGY	TUMOR MARKER	AFB/PCR/MYCOBAC	EXUDATIVE P.E	P.E		TB EMPYEMA		PROBABLE	CONFIRMED	SIMPLE	COMPLEX	TYPICAL	BORDERLINE	OMPLICATED	COMPLICATED	.
						U/L	MG/DL						PROBABLE	CONFIRMED	DEFINITIVE	PROBABLE									
ND	NEG	NEG	NEG	NEG	POS	16	N	N	N	ND	ND	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	20.3	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	211.8	N	N	N	ND	NEG	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
ND	NEG	GPC	NEG	NEG	NEG	37	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	NEG	NEG	42.2	N	TICA	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	198	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	79	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	GNB	NEG	P.AERU	NEG	23	N	N	N	ND	ND	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	67	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	K.PNEUM	NEG	18.62	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	170	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	182	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
20.2	NEG	NEG	NEG	NEG	NEG	48	N	PCV- 39.2	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	176	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	122	N	N	N	ND	ND	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	158	N	N	LN- GRANULOMATOUS DS	ND	ND	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	109	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	ACINETOB	NEG	154	N	N	N	ND	NEG	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	NEG	NEG	200	N	COVID RTP	N	ND	NEG	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	K.PNEUM	NEG	43.87	N	N	N	ND	NEG	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	NEG	NEG	220	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	POS	NEG	POS	NEG	NEG	99	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	POS	35.2	N	N	N	ND	ND	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	88	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	18.5	N	N	CHRONIC	NEG	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	74.57	N	N	N	NEG	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	MRSA	NEG	115	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	109	N	N	N	ND	ND	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	56	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	101	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	198	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	MRSA	NEG	34	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	30	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	28	N	N	N	NEG	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	171	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	182	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	POS	NEG	NEG	NEG	NEG	158	N	N	LN- TB	ND	ND	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	38	N	N	N	ND	ND	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
ND	NEG	NEG	NEG	MRSA	NEG	67	N	N	CHRONIC	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	NEG	NEG	24	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	200	N	N	N	ND	POS	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	POS	45	N	N	ADENOCA	ND	ND	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	70	N	N	N	ND	NEG	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	65	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N

HEMOTHORAX	PULM.EMBOLISM	VIRAL	PARASITIC EMPYEMA	UNKNOWN	SPECIFIC TO ETIOLOGY		INTERVENT	TREATMENT							OUTCOME											
					ORGANISM	CARCINOMA		ABX	ATT	CHEMO	RADIO	SX	DELAY	OTHER	RESOLUTION OF SYMPTOMS			DAMA/EXPIRY			F/U CHEST XRAY			F/U CT CHEST		DX CHANGE
															AT D/C	1 MONTH	3 MONTH	AT D/C	1 MONTH	3 MONTH	AT D/C	1 MONTH	3 MONTH	1 MONTH	3 MONTH	1 MONTH
N	N	N	N	N	N	CA LUNG	PCD	Y	N	N	N	N	155 DAYS	N	N	NA	NA	DAMA	EXP	EXP	SIGNFCNT	NA	NA	NA	NA	NA
N	Y	N	N	N	N	N	PCD	Y	N	N	N	N	90 DAYS	LMWH	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	N	N	PCD	Y	N	N	N	N	25 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	BACTERIAL	N	PCD	Y	N	N	N	N	16 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	Y	N	TICA	N	PCD	Y	N	N	N	N	17 DAYS	RIALS	Y	Y	Y	NA	NA	NA	RES	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD	Y	Y	N	N	N	20 DAYS	STRD	Y	Y	Y	NA	NA	NA	MILD	MILD	MINIMAL	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	35 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	RES	RES	N	N	N
N	N	N	N	N	P.AERU	N	PCD	Y	N	N	N	N	127 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	55 DAYS	N	N	Y	Y	NA	NA	NA	MOD	MILD	RES	N	N	N
N	N	N	N	N	K.PNEUM	N	PCD + STK	Y	N	N	N	N	4DAYS	N	N	N	Y	NA	NA	NA	MOD	MOD	MILD	N	N	N
N	N	N	N	N	TB	N	N	Y	Y	N	N	N	12 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	365 DAYS	STRD	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
Y	N	N	N	N	N	N	ICD	Y	N	N	N	VATS	10 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	P THICK	P THICK	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	25 DAYS	STRD	N	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD	Y	Y	N	N	N	36 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD + STK	Y	Y	N	N	N	29DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	33 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	P THICK	N	N	N
N	N	N	N	N	ACINETOBACTER	N	PCD	Y	N	N	N	N	15 DAYS	N	DAMA	NA	NA	DAMA	EXP	EXP	PCD IN SITU	NA	NA	NA	NA	NA
N	N	Y	N	N	N	N	N	Y	N	N	N	N	7 DAYS	ANTIVIRAL	Y	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	N	K.PNEUM	N	PCD + STK	Y	N	N	N	N	5 DAYS	N	Y	Y	LTF	NA	NA	NA	RES	RES	LTF	N	NA	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	10 DAYS	N	N	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	20 DAYS	N	N	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	N	CA OVARY	N	Y	N	Y	N	N	7 DAYS	N	Y	NA	NA	NA	EXP	EXP	MOD	NA	NA	NA	NA	NA
N	N	N	N	N	TB	N	PCD	N	Y	N	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	P THICK	P THICK	N	N	N
N	N	N	N	Y	N	N	PCD	Y	N	N	N	N	NO DEFINIT	N	Y	N	Y	NA	NA	NA	MILD	RECURREN	P THICK	N	N	N
N	N	N	N	N	L	N	PCD + STK	Y	N	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	RES	RES	RES	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	Y	N	N	N	N	18 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD	N	Y	N	N	N	64 DAYS	N	N	Y	Y	NA	NA	NA	MILD	MILD	P THICK	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	14 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	22 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	20 DAYS	ATT	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	N	N	N	N	N	18 DAYS	N	Y	Y	Y	NA	NA	NA	RES	RES	RES	N	N	N
N	N	N	N	Y	N	N	N	Y	N	N	N	N	NO DEFINIT	N	Y	N	N	NA	NA	NA	MILD	RECURREN	MOD	LYMPHANG	N	M.P.E
N	N	N	N	Y	N	N	N	Y	N	N	N	N	NO DEFINIT	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	100 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	7 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD	N	Y	N	N	N	40 DAYS	STRD	Y	Y	Y	NA	NA	NA	MILD	P THICK	P THICK	N	N	N
N	N	N	N	N	N	N	PCD	Y	N	N	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	MRSA	N	ICD	Y	N	N	N	VATS	2 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	P THICK	P THICK	N	N	N
N	Y	N	N	N	N	N	N	Y	N	N	N	N	3 DAYS	LMWH	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD + STK	Y	Y	N	N	N	15 DAYS	STRD	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	N	CA LUNG	N	N	Y	N	N	N	26 DAYS	N	Y	Y	LTF	NA	NA	LTF	MILD	MILD	LTF	N	NA	N
N	N	N	N	N	N	N	PCD	Y	N	N	N	N	30 DAYS	PES	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	TB	N	N	Y	Y	N	N	N	12 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N

E	COMPLICATION			TREATMENT CHANGE	
3 MONTH	AT D/C	1 MONTH	3 MONTH	1 MONTH	3 MONTH
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	PCD OUT
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	PCD OUT	N
N	N	N	N	PCD OUT	N
N	N	HEPATITIS	CHEST PAI	MOD ATT	N
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
NA	N	N	LTF	N	LTF
N	N	N	N	N	N
N	N	N	N	N	N
NA	N	NA	NA	NA	NA
N	H.PNEU	N	N	N	N
N	N	RECURRE	N	VATS	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	HEPATITIS	N	MOD ATT	N
N	N	HEPATITIS	N	MOD ATT	N
N	N	HEPATITIS	N	MOD ATT	N
N	N	AKI	N	MOD ATT	N
N	N	N	N	N	N
N	N	RECURRE	N	CHEMOTH	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
NA	NA	NA	NA	NA	NA
NA	N	N	LTF	N	LTF
NA	NA	NA	NA	N	NA
N	N	N	N	N	N

S.NO	NAME	MRD	AGE	GENDER	S/E STATUS	OCCUPATION	DURATION OF HOSPITALISATION	PRESENT HISTORY									DURATION	PAST HSITORY									PERSONAL HISTORY				OTHER	BMI
							DAYS	CHEST PAIN	SOB	FEVER	TRAUMA	COUGH	EXPECTORATION	HEMPOTYSIS	OTHER	DAYS	TB	HTN	DM	CLD	CKD	PLHIV	HCV	HBV	OTHER	SMOKE	ALCOHOL	MENSTRUAL HISTORY		KG/M2
45	PK	936135	33	F	UMC	D	10	Y	N	N	N	Y	N	N	N	5	N	N	N	N	N	N	N	N	N	N	N	IR	N	23.4
46	MS	668150	41	M	LOWER	FA	9	N	N	N	N	N	N	Y	N	60	Y	N	N	N	N	N	Y	N	N	Y	Y	NA	N	21.8
47	SK	921739	25	F	UPPER	D	8	Y	Y	N	N	Y	N	N	WT LOSS	12	N	N	N	N	N	N	N	N	N	N	R	N	24.5
48	OP	925030	80	M	UMC	D	16	N	Y	Y	N	Y	N	N	PAIN ABDMN	15	N	N	N	N	N	N	N	N	N	N	NA	N	28
49	GDS	977916	71	M	UPPER	D	27	Y	Y	Y	N	Y	N	N	N	15	N	N	N	N	N	N	N	N	N	N	N	NA	N	25.9
50	DS	976814	24	M	UMC	D	9	N	Y	Y	N	Y	Y	N	N	6	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.8
51	GK	949288	56	M	LMC	DRIVER	10	Y	N	Y	N	Y	N	N	N	5	N	N	N	N	N	N	N	N	N	N	N	NA	N	24.4
52	JS	885575	44	M	UMC	FA	45	N	Y	Y	N	Y	Y	N	N	5	N	N	N	N	N	N	N	N	CA LUNG	N	N	NA	N	23.8
53	RJ	982326	76	M	UPPER	B	14	N	Y	Y	N	Y	N	N	N	10	N	N	N	N	N	N	N	N	N	N	Y	NA	N	19.8
54	AK	980747	32	M	UMC	B	20	Y	N	Y	Y	N	N	N	N	10	Y	N	N	N	N	N	N	N	N	N	Y	NA	N	24.8
55	NS	981856	62	M	UMC	FA	5	N	Y	Y	N	Y	Y	N	N	20	N	N	Y	N	N	N	N	N	N	N	N	NA	N	24.5
56	SS	981789	49	M	UMC	SERVICEMAN	13	Y	Y	N	N	N	N	N	N	13	N	N	N	N	N	N	N	N	N	N	Y	NA	N	22.9
57	SA	981143	30	M	UMC	B	10	Y	N	N	N	Y	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	NA	N	19.9
58	DK	979817	40	M	UMC	FA	8	N	Y	N	N	Y	Y	Y	N	15	N	N	N	N	N	N	N	N	N	N	N	NA	N	24.8
59	RK	723431	24	F	UMC	HCW	3	Y	N	Y	Y	N	N	N	N	20	N	N	N	N	N	N	N	N	N	N	N	R	N	21.3
60	SB	979956	34	F	UMC	D	10	N	N	Y	N	Y	N	N	N	12	N	Y	N	N	N	N	N	N	N	N	N	R	N	26.5
61	A	963597	21	M	UPPER	D	12	Y	Y	N	N	N	N	N	N	120	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.8
62	RKG	979820	52	M	UMC	B	25	N	Y	N	N	Y	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	NA	N	23.3
63	AV	969733	65	M	UMC	B	36	N	Y	N	N	N	N	N	N	12	N	Y	Y	N	N	N	N	N	N	N	N	NA	N	26.5
64	IS	928766	55	M	LMC	FA	5	N	Y	Y	N	N	N	N	WT LOSS	7	N	N	Y	Y	N	N	N	N	N	N	N	NA	N	24.7
65	PK	936135	33	F	UMC	D	7	N	Y	N	N	N	N	N	N	12	N	N	N	N	N	N	N	N	N	N	N	R	N	25.5
66	S	947895	56	M	UMC	DRIVER	5	N	Y	Y	N	N	N	N	N	15	N	N	N	N	N	N	N	N	N	N	N	NA	N	20.8
67	DS	991673	66	M	UMC	B	7	Y	Y	N	N	N	N	N	N	25	N	Y	Y	N	N	N	N	N	CAD	N	N	NA	N	23.8
68	R	620858	64	M	UMC	B	10	Y	Y	Y	N	Y	Y	N	N	20	Y	N	Y	N	N	N	N	N	N	Y	Y	NA	N	22.9
69	GK	940300	65	F	LOWER	D	5	N	Y	Y	N	N	N	N	N	30	Y	N	N	N	N	N	N	N	N	N	N	PM	N	19.9
70	SKA	592481	62	M	LMC	FA	28	Y	Y	Y	N	Y	Y	N	N	17	N	Y	Y	N	N	N	Y	N	N	N	N	NA	N	17.5
71	RS	1128967	24	M	UPPER	D	60	Y	Y	Y	Y	Y	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	NA	N	22
72	NK	1127718	70	F	UMC	D	12	Y	Y	Y	N	N	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	PM	N	24.6
73	GS	1009083	32	M	UMC	B	7	N	Y	N	N	N	N	N	N	5	N	N	N	N	N	N	N	N	HL	Y	N	NA	N	22.1
74	ND	1063147	65	F	UMC	D	5	Y	Y	Y	N	N	Y	N	N	5	N	Y	Y	N	N	N	N	N	N	N	N	PM	N	28.9
75	KKB	300231	22	F	UMC	D	5	Y	N	N	N	N	N	N	N	15	N	N	N	N	N	N	N	N	N	N	N	R	N	18.8
76	RRP	895518	71	M	UPPER	D	10	Y	Y	Y	N	N	N	Y	N	20	N	Y	N	N	N	N	N	N	N	N	N	NA	N	24.7
77	GS	1027231	70	M	UMC	D	12	N	Y	Y	N	N	N	N	WT LOSS	15	Y	N	Y	N	N	N	N	N	N	N	N	NA	N	21.7
78	SS	398201	48	M	UMC	B	20	N	Y	N	N	N	Y	N	N	20	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.9
79	SL	1020779	83	M	UPPER	B	9	N	Y	N	N	Y	N	N	N	16	N	N	N	N	N	N	N	N	N	N	N	NA	N	26.3
80	KS	951772	75	M	UMC	D	11	N	Y	Y	N	N	N	N	WT LOSS	13	N	N	Y	N	N	N	N	N	BA	N	N	NA	N	19.9
81	PD	979763	36	F	UMC	D	7	N	N	Y	N	Y	N	N	N	15	N	N	N	N	N	N	N	N	N	N	N	R	N	22.6
82	BK	979743	40	F	UPPER	D	8	N	Y	Y	N	Y	N	N	N	20	N	N	Y	N	N	N	N	N	N	N	N	R	N	26.5
83	RK	979731	52	M	UMC	B	16	N	N	Y	Y	N	Y	N	N	10	N	N	N	N	N	N	N	N	N	Y	Y	NA	N	20.22
84	DK	979817	40	M	LOWER	DWW	5	Y	Y	N	N	N	N	N	N	7	N	N	N	N	N	N	N	N	N	Y	Y	NA	N	18.9
85	DS	979151	65	M	LMC	B	7	Y	Y	N	N	N	N	N	N	10	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.6
86	VL	1016193	38	F	UMC	D	20	N	Y	Y	Y	N	N	N	N	5	N	N	N	N	N	N	N	N	N	N	N	AMENORRHEA	N	25.4
87	SS	981789	85	M	UPPER	D	14	N	Y	Y	N	Y	N	N	N	22	N	Y	Y	Y	N	N	N	N	N	N	N	NA	N	23.8
88	A	389235	35	M	UMC	B	5	Y	N	N	Y	Y	N	N	N	5	N	N	N	N	N	N	N	N	N	N	N	NA	N	24.9
89	ML	1001372	54	M	UMC	FA	10	Y	N	Y	N	Y	N	N	N	7	N	N	N	N	N	N	N	N	N	N	N	NA	N	25.4
90	DS	991673	47	M	LOWER	FA	13	Y	N	N	N	N	N	N	N	12	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.4
91	K	980270	56	M	UMC	B	12	Y	Y	N	N	Y	N	N	N	3	N	N	N	N	N	N	N	N	N	N	N	NA	N	28.9

SPO2 LESS THAN 94 RECQUIRING OXYEGEN	ICTERUS	CLUBBING	LAP	CHEST					CVS	CNS	ABD	OTHER	HB	TLC	DLC	CREAT	ALBUMIN	PROTEIN	OT	PT	ALP	ESR	S .LDH	MANTOUX	CXR				
				SYMMETRY	TRACHEA	TACTILE FREMITUS	PERCUSSION	AUSCULTATION																	EFFUSION	LATERALITY	OPACITY	M.SHIFT	OTHERS
													G/DL	10^3/UL	NE/L/E	MG/DL	G/DL	G/DL	U/L	U/L	U/L		U/L	MM					
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.1	10	NE	0.67	3.04	5.9	91	45	329	89	423	NEG	Y	RT	N	Y	N
Y	N	Y	N	AS	S	DECREASED	DULL	B/L ABSENT	NAD	NAD	NAD	NAD	14	7.3	NE	0.74	4.18	7.3	179	248	139	11	253	ND	Y	B/L	Y	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.8	8	L	0.77	4.1	6.9	56	78	129	79	295	POS	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	TENDER	NAD	7.6	4.5	NE	0.89	2.09	4.1	35	13	388	98	291	NEG	Y	RT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.6	6.2	L	0.47	3.29	7.2	17	27	91	120	200	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	9.8	15.5	NE	0.81	3.05	5.9	35	15	167	72	412	NEG	Y	LT	N	Y	MILLIARY M
N	N	N	N	S	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.9	4.5	L	0.99	4.1	6.9	24	13	56	77	178	NEG	Y	LT	N	N	CAVITY
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.1	7.7	L	1.3	3.3	6.5	26	29	119	89	300	NEG	Y	RT	Y	Y	N
Y	N	N	N	AS	C	DECREASED	DULL	B/L ABSENT	NAD	NAD	NAD	NAD	14.8	30	NE	2.8	2.8	6.6	88	70	120	48	203	NEG	Y	B/L	Y	Y	N
Y	N	N	N	AS	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.4	40.3	NE	2.65	1.9	4.6	144	48	234	39	521	NEG	Y	LT	N	Y	FIBROSIS
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.1	20	NE	1.12	3.8	7.2	48	49	126	48	193	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.8	15.1	NE	0.94	2.8	5.6	67	34	111	120	ND	ND	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11	9.7	NE	0.6	4.08	5.7	89	52	127	50	665	NEG	Y	LT	N	N	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	9	24	NE	0.39	2.4	6.3	178	80	145	90	233	NEG	Y	LT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	11.2	5.4	L	0.88	3.1	5.1	101	34	111	45	159	NEG	Y	RT	N	N	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.1	11.2	NE	0.55	2.4	5.6	68	45	120	58	200	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	15.1	10.6	L	1.1	2.9	5.6	77	60	77	12	150	NEG	Y	LT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.9	13.3	NE	1.8	3.3	4	45	70	155	17	550	NEG	Y	LT	Y	Y	FIBROSIS
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.6	16	NE	2.1	2.45	4.8	50	136	78	22	344	NEG	Y	LT	Y	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.9	12.1	NE	0.98	3.54	6.89	89	67	110	18	120	NEG	Y	RT	N	N	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.9	13	L	1.06	3.98	7	44	55	67	45	130	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.1	10	L	0.56	3.2	6.97	55	45	89	76	198	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.4	8.8	L	0.65	3.05	5.2	23	16	43	16	110	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	13.4	6.7	L	0.88	2.11	5.1	45	34	67	88	210	NEG	Y	LT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.8	18	L	2.11	3.31	7.3	141	61	155	12	387	NEG	Y	RT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	ENCEPH	NAD	NAD	8.6	19.8	NE	3.8	2.1	5.1	200	214	168	44	340	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.8	10.9	NE	0.55	2.41	6.3	27	29	56	46	172	NEG	Y	RT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	8.8	9	NE	1.2	2.89	6.7	78	88	76	67	154	NEG	Y	RT	Y	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	B/L ABSENT	NAD	NAD	NAD	NAD	11.2	5	NE	0.98	3.1	5.6	34	67	98	45	198	NEG	Y	B/L	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	ENCEPH	ASCITIS	NAD	8.8	26	NE	3.4	2.56	5.8	98	67	123	56	223	ND	Y	RT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.2	10	NE	0.98	4.1	7.4	32	23	67	86	121	NEG	Y	LT	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	13.1	12.1	NE	3.1	2.8	5.6	56	55	89	78	111	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.1	14.9	NE	1.9	2.7	4.9	55	45	75	46	142	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.8	11.1	NE	0.98	4.1	6.4	23	22	45	56	110	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	11	7.8	NE	1.01	3.98	6.2	23	32	35	76	132	NEG	Y	RT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	9.8	27.2	NE	0.58	2.71	5.8	29	29	173	64	157	NEG	Y	LT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	13	9.4	NE	0.57	3.12	6.1	28	23	117	9	120	NEG	Y	RT	N	N	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.2	20	NE	0.38	2.1	4.8	49	59	156	46	150	NEG	Y	RT	N	N	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	14.1	12.8	NE	0.56	3.42	6.67	55	45	119	65	117	NEG	Y	RT	Y	N	N
N	N	N	N	S	C	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.9	16	NE	1.12	3.78	6.7	45	44	167	79	250	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.5	11	NE	0.97	3.4	7.5	34	22	65	78	112	NEG	Y	LT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	7.5	29	NE	5.7	2.8	5.8	88	110	210	65	210	NEG	Y	RT	N	Y	N
Y	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	10.8	14.9	L	0.67	3.25	5.74	24	43	76	93	173	NEG	Y	RT	N	Y	N
N	N	N	N	S	C	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.1	10	L	0.82	4.8	6	35	45	88	54	185	NEG	Y	RT	N	N	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	14.1	12.1	L	2.1	3.4	5	55	54	79	98	121	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12.1	9.8	L	2.23	3.3	5.8	55	65	127	119	202	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASED	DULL	LT ABSENT	NAD	NAD	NAD	NAD	11.9	5	E	1.14	2.8	6.2	33	34	69	45	191	NEG	Y	LT	N	Y	N

USG CHEST						CT CHEST	USG P.TAP		COMPLICATIONS			PLEURAL FLUID ANALYSIS													
LATERALITY	MILD	MOD	SIGNIFICANT	MULTIPLE LOCULATI	ON-SINGLE		DIAGNOSTIC TAP	THERAPEUTIC TAP	Y/N	HEMOTHORAX	H.PNEUMOTHORAX	HEMORRHAGIC	STRAW	PUS	OTHER	CHYLOUS	PROTEIN	GLUCOSE	LDH	PH	TLC	L	NE	L:NE	RBC
																	G/DL	MG/DL	U/L		CELLS/MM3	%	%		CELLS/MM3
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	3.1	72	2600	7.5	400	80	20	4	10000
B/L	N	Y	N	N	N	CA LUNG WITH MOD B/L P.E	Y	Y	N	N	N	N	Y	N	N	N	4.4	96	206	ND	250	70	30	2.3	250
LT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.4	68	261	7.3	200	90	10	9	100
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	N	N	ANCHOVY.S	N	2.6	114	271	ND	10	0	100	0	0
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	4.4	123	262	7.4	1000	80	20	4	100
LT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	N	Y	N	N	5.5	3	4006	ND	200	60	40	1.5	100
LT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.3	78	222	ND	300	75	25	3	90
RT	N	Y	N	N	N	CA LUNG WITH MOD RT P.E	Y	N	N	N	N	Y	N	N	N	N	3.8	99	550	ND	600	90	10	9	1200
B/L	N	Y	N	Y	N	ND	Y	N	N	N	N	N	Y	N	N	N	5	10	650	7.2	900	20	80	0.25	1400
LT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.8	1	1444	6.5	1000	20	80	0.25	0
LT	N	Y	N	N	Y	ND	Y	N	N	N	N	N	Y	N	N	N	4.8	52	800	7.2	600	30	70	0.42	200
LT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	5	2	ND	ND	10000	10	90	0.1	200
LT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.5	91	747	ND	2200	70	30	2.3	20000
LT	N	N	Y	N	Y	ND	Y	N	N	N	N	N	Y	N	N	N	4.4	1	1027	ND	3800	10	90	0.1	100
RT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	2.9	79	190	ND	100	80	20	4	20
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.7	67	225	ND	500	70	30	2.3	1200
LT	N	Y	N	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	3.4	40	687	ND	2000	60	40	1.5	10000
LT	N	Y	N	N	N	EXTENSIVE CCC - SCRAD C WITH EARLY FIBROSIS WITH	Y	N	N	N	N	N	Y	N	N	N	2.2	103	889	ND	7600	20	80	0.25	20000
LT	N	Y	N	N	Y	LT LOCULATED P.E	Y	N	N	N	N	N	Y	N	N	N	3.5	31	1587	7	300	80	20	4	100
RT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.1	88	300	ND	450	90	10	9	300
LT	N	Y	N	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	3.5	66	450	ND	500	70	30	2.3	250
RT	N	N	Y	N	N	P.E	Y	Y	N	N	N	N	Y	N	N	N	5.3	169	457	ND	700	80	20	4	100
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	2.6	205	123	ND	300	100	0		0
LT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.4	89	356	ND	700	90	10	9	80
RT	N	Y	N	Y	N	ND	Y	Y	N	N	N	N	Y	N	N	N	5.1	2	405	ND	1600	30	70	0.42	10000
LT	N	Y	N	N	Y	ND	Y	N	N	N	N	N	N	Y	N	N	3.3	11	1216	ND	100	90	10	9	100
RT	N	Y	N	N	N	SIGNIFICANT RT P.E	Y	N	N	N	N	Y	N	N	N	N	4.8	66	672	ND	500	85	15	5.6	350000
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.8	88	230	ND	500	70	30	2.3	90
B/L	N	N	Y	N	N	SIGNSCNT B/L P.E	Y	Y	N	N	N	N	N	N	N	Y	2.9	65	221	ND	100	55	45	1.2	0
RT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	3.6	22	876	ND	400	10	90	0.1	200
LT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.5	88	234	ND	450	85	15	5.6	70
LT	N	Y	N	N	Y	ND	Y	N	N	N	N	N	Y	N	N	N	4.1	23	760	ND	400	30	70	0.42	100
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.1	33	900	ND	990	10	90	0.1	500
RT	N	Y	N	N	N	MOD RT P.E	Y	Y	N	N	N	N	Y	N	N	N	3.3	78	180	ND	100	45	55	0.8	10
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.4	70	110	ND	100	55	45	1.2	110
LT	N	Y	N	N	N	LT LUNG MASS + MOD LT P.E	Y	N	N	N	N	N	Y	N	N	N	4.3	176	183	ND	300	100	0		100
RT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.3	120	182	ND	450	60	40	1.5	2000
RT	Y	N	N	N	Y	ND	Y	N	N	N	N	N	N	Y	N	N	4.8	40	202	ND	400	20	80	0.25	500
RT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.9	90	220	ND	500	10	90	0.1	100
LT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	5.6	20	1000	ND	600	20	80	0.25	300
LT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.3	97	200	ND	100	45	55	0.8	80
RT	N	Y	N	N	N	ND	Y	N	N	N	N	Y	N	N	N	N	5.8	77	890	ND	500	90	10	9	10000000
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	4.5	86	189	ND	300	70	30	2.3	200
RT	Y	N	N	N	N	ND	Y	N	N	N	N	Y	N	N	N	N	3.9	94	199	ND	100	90	10	9	1200
LT	N	N	Y	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	2.9	39	397	ND	90	40	60	0.6	150
RT	N	Y	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	3.1	78	210	ND	200	90	10	9	210
LT	N	Y	N	N	N	PULMONARY EMBOLISM WITH MOD LT P.E	Y	N	N	N	N	Y	N	N	N	N	3.3	99	300	ND	100	90	10	9	20000

												DX	TB				M. P.E		EMPYEMA		PP.E				CHYLOTHORAX
PCV	AFB	GRAM STAIN	CBNAAT	C/S	M CELL	ADA	TG	OTHER	HISTOPATHOLOGY	TUMOR MARKER	AFB/PCR/MYCOBAC	EXUDATIVE P.E	P.E		TB EMPYEMA		PROBABLE	CONFIRMED	SIMPLE	COMPLEX	TYPICAL	BORDERLINE	OMPLICATED	COMPLICATED	.
						U/L	MG/DL						PROBABLE	CONFIRMED	DEFINITIVE	PROBABLE									
ND	NEG	NEG	NEG	NEG	NEG	21	N	N	N	POS	ND	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	POS	46	N	N	ADENOCA	POS	ND	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	96	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	14.8	N	E.HISTOLY	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	87	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	366	N	N	N	ND	NEG	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	98	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	POS	56	N	N	N	ND	NEG	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	GPC	NEG	MRSA	NEG	54	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	S.PNEUM/	NEG	232	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	GNC	NEG	E.COLI	NEG	42	N	N	N	ND	NEG	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	MRSA	NEG	361	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	287	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	185	N	N	N	ND	NEG	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	NEG	NEG	79	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	95	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	BURKHOLI	NEG	18.3	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	NEG	NEG	39	N	COVID RTP	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	K.PNEUM	NEG	178	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	NEG	NEG	120	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	56	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	POS	87	N	N	ADENOCA	ND	ND	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	64.4	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	184	N	N	N	ND	NEG	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	70	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	ACINETOB	NEG	78.8	N	N	N	ND	NEG	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
16.4	NEG	NEG	NEG	NEG	NEG	161	N	PCV- 34.9	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	200	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	34	240	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	Y
ND	NEG	NEG	NEG	NEG	NEG	34	N	SPUTUM- E	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	90	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	54	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	E.COLI	NEG	34	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	NEG	NEG	45	N	N	N	ND	NEG	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	112	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	28.7	N	N	NSCC	ND	ND	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N
ND	NEG	NEG	POS	NEG	NEG	289	N	N	N	ND	ND	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	GNC	NEG	E.COLI	NEG	10.2	N	N	N	ND	ND	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	23	N	SPUTUM-K	N	ND	NEG	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N
ND	NEG	NEG	NEG	MRSA	NEG	37	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	32	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
15.8	NEG	NEG	NEG	NEG	NEG	45	N	PCV- 29.8	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	87	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
4	NEG	NEG	NEG	NEG	NEG	34	N	PCV-38.2	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	E.COLI	NEG	45	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N
ND	NEG	NEG	NEG	NEG	NEG	89	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	44	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N

HEMOTHORAX	PULM.EMBOLISM	VIRAL	PARASITIC EMPYEMA	UNKNOWN	SPECIFIC TO ETIOLOGY		INTERVENT	TREATMENT							OUTCOME											
					ORGANISM	CARCINOMA		ABX	ATT	CHEMO	RADIO	SX	DELAY	OTHER	RESOLUTION OF SYMPTOMS			DAMA/EXPIRY			F/U CHEST XRAY			F/U CT CHEST		DX CHANGE
															AT D/C	1 MONTH	3 MONTH	AT D/C	1 MONTH	3 MONTH	AT D/C	1 MONTH	3 MONTH	1 MONTH	3 MONTH	1 MONTH
N	N	N	N	N	N	UNKNOWN	PCD	Y	N	N	N	N	7 DAYS	N	N	LTF	LTF	DAMA	LTF	LTF	NA	NA	NA	NA	NA	NA
N	N	N	N	N	N	CA LUNG	PCD	Y	N	Y	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	MILD	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	MILD	N	N	N
N	N	N	Y	N	E.HISTOLYTICA	N	PCD	Y	N	N	N	N	15 DAYS	ANTIMALRIALS	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	17 DAYS	N	Y	Y	Y	NA	NA	NA	RES	RES	RES	N	N	N
N	N	N	N	N	TB	N	PCD	Y	Y	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	P THICK	P THICK	N	N	N
N	N	N	N	N	TB	N	N	N	N	N	N	N	7 DAYS	N	N	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	N	CA LUNG	N	Y	N	Y	N	N	50 DAYS	N	Y	Y	N	NA	NA	NA	MOD	MOD	RECURREN	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	Y	N	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	.AERU	N	ICD + STK	Y	N	N	N	N	10 DAYS	PES	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	E.COLI	N	PCD	Y	N	N	N	N	22 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	Y	N	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	RES	RES	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	12 DAYS	N	Y	N	N	NA	NA	NA	PCD IN SITU	RECURREN	RECURREN	ANTR. MED	N	M.P.E
N	N	N	N	N	N	N	ICD	Y	N	N	N	N	NO DEFINIT	N	Y	N	N	NA	NA	NA	MILD	MILD	MILD	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	24 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	P THICK	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	DERIA	N	ICD	Y	N	N	N	N	130 DAYS	N	Y	N	N	NA	NA	NA	MOD	MOD	MOD	L.MOD P.E	N	UNKOWN
N	N	Y	N	N	COVID	N	N	Y	N	N	N	N	15 DAYS	ANTIVIR	Y	N	NA	NA	NA	EXP	MILD	MILD	NA	N	NA	N
N	N	N	N	N	K.PNEUM	N	PCD	Y	N	N	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	MILD	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	P THICK	P THICK	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	17 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	N	CA LUNG	N	Y	N	Y	N	N	20 DAYS	N	Y	N	N	NA	NA	NA	MOD	MOD	RECURREN	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	30 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	RES	RES	N	N	N
N	N	N	N	N	TB	N	N	Y	N	N	N	N	25 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	MILD	N	N	N
N	N	N	N	N	N	N	PCD + STK	Y	Y	N	N	N	EXP	PES	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	ACTER	N	PCD	Y	N	N	N	N	25 DAYS	PES	N	NA	NA	NA	EXP	EXP	MOD	NA	NA	N	NA	NA
Y	N	N	N	N	N	N	ICD	Y	N	N	N	VATS	10 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	P THICK	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	12 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	RES	N	N	N
N	N	N	N	N	N	N	ICD	N	N	Y	N	N	5 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	MILD	N	N	N
N	N	N	N	N	N	N	PCD	Y	N	N	N	N	EXP	PES	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	TB	N	N	N	Y	N	N	N	15 DAYS	N	N	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	N	N	PCD	Y	N	N	N	N	22 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	MILD	N	N	N
N	N	N	N	N	E.COLI	N	PCD	Y	N	N	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	Y	N	N	N	Y	N	N	N	N	NO DEFINIT	N	Y	Y	N	NA	NA	NA	MILD	MILD	RECURREN	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	21 DAYS	N	N	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	N	N	CA LUNG	N	Y	N	Y	N	N	20 DAYS	N	Y	Y	NA	NA	NA	EXP	MILD	MILD	NA	N	NA	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	25 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	MILD	N	N	N
N	N	N	N	N	E.COLI	N	PCD	Y	N	N	N	N	20 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	N	N	N	N	Y	N	N	N	N	11 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	MILD	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	Y	N	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	P THICK	N	N	N
N	N	N	N	Y	N	N	N	Y	N	N	N	N	NO DEFINIT	N	Y	N	N	NA	NA	NA	MOD	MOD	RECURREN	N	MOD LT P.E	N
Y	N	N	N	N	N	N	ICD	Y	N	N	N	N	EXP	N	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	TB	N	N	N	Y	N	N	N	24 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	Y	N	N	N	Y	N	N	N	N	NO DEFINIT	N	Y	Y	Y	NA	NA	NA	MILD	P THICK	P THICK	N	N	N
N	N	N	N	N	E.COLI	N	PCD	Y	N	N	N	N	10 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	MILD	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	14 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	Y	N	N	N	N	N	N	Y	N	N	N	N	5 DAYS	LMWH	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N

E	COMPLICATION			TREATMENT CHANGE	
3 MONTH	AT D/C	1 MONTH	3 MONTH	1 MONTH	3 MONTH
NA	NA	LTF	LTF	LTF	LTF
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	HEPATITIS	N	MOD ATT	N
N	N	N	N	N	N
N	N	N	N	N	TH. P. TAP
N	N	N	N	N	N
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
N	N	N	N	N	N
N	N	RECURRE	RECURREN	CHEMOTH	N
N	N	N	N	N	N
N	N	HEPATITIS	N	MOD ATT	N
N	N	N	N	N	N
N	N	N	N	N	N
NA	N	N	NA	N	NA
N	N	N	N	N	N
N	N	CHEST PA	N	N	N
N	N	N	N	N	N
N	N	N	METS TO B	N	TH. P. TAP
N	N	N	N	N	N
N	N	N	N	N	N
NA	NA	NA	NA	NA	NA
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
NA	N	N	COVID POS	N	NA
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	RECURREN	N	PCD
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
N	N	HEPATITIS	N	MOD ATT	N
N	N	N	N	N	N

S.NO	NAME	MRD	AGE	GENDER	S/E STATUS	OCCUPATION	DURATION OF HOSPITALISATION	PRESENT HISTORY									DURATION	PAST HSITORY										PERSONAL HISTORY			OTHER	BMI
							DAYS	CHEST PAIN	SOB	FEVER	TRAUMA	COUGH	EXPECTORATION	HEMPOTYSIS	OTHER	DAYS	TB	HTN	DM	CLD	CKD	PLHIV	HCV	HBV	OTHER	SMOKE	ALCOHOL	MENSTRUAL HISTORY		KG/M2
92	CS	1140682	48	M	LMC	FA	18	Y	Y	Y	N	Y	N	N	N	30	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.8
93	K	980270	38	F	LMC	D	5	N	Y	Y	N	N	N	N	N	20	N	N	N	N	N	N	N	N	HYSTERECTOMY	N	N	IR	N	18.9
94	SK	1159639	67	M	UMC	D	10	N	Y	Y	N	Y	N	N	N	18	N	Y	N	N	N	N	N	N	N	N	Y	NA	N	25.9
95	KS	1136181	56	M	UPPER	B	5	Y	Y	Y	N	Y	Y	N	N	3	N	Y	Y	N	N	N	N	N	N	N	N	NA	N	24
96	GDS	977916	34	M	LMC	FA	7	Y	N	N	N	N	N	N	N	7	N	N	N	N	N	N	N	N	N	N	N	NA	N	22.1
97	BR	154462	46	M	UMC	B	10	Y	Y	N	N	N	N	N	AP LOSS	12	Y	Y	N	N	N	N	N	N	N	N	NA	N	28.9
98	SK	1009498	59	M	UMC	B	7	Y	Y	N	N	Y	N	N	N	10	N	Y	Y	N	N	N	N	N	N	N	N	NA	N	24
99	SK	1002715	56	M	UMC	B	10	Y	Y	N	N	N	N	N	N	5	N	N	N	N	N	N	N	N	N	N	Y	NA	N	23
100	JS	1023754	25	M	LMC	DWW	5	N	Y	Y	N	Y	Y	N	WT LOSS	30	N	N	N	N	N	N	N	N	N	Y	N	NA	N	18.9

SPO2 LESS THAN 94 RECQUIRING OXYEGEN	ICTERUS	CLUBBING	LAP	CHEST					CVS	CNS	ABD	OTHER	HB	TLC	DLC	CREAT	ALBUMIN	PROTEIN	OT	PT	ALP	ESR	S .LDH	MANTOUX	CXR				
				SYMMETRY	TRACHEA	TACTILE FREMITUS	PERCUSSION	AUSCULTATION																	EFFUSION	LATERALITY	OPACITY	M.SHIFT	OTHERS
													G/DL	10^3/UL	NE/L/E	MG/DL	G/DL	G/DL	U/L	U/L	U/L		U/L	MM					
N	N	N	N	AS	S	DECREASE	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10.8	11.1	L	0.9	2.3	5	65	55	128	88	201	NEG	Y	LT	N	Y	N
N	N	N	N	AS	S	DECREASE	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.8	7.7	L	0.99	3.6	5.8	35	43	112	76	540	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASE	DULL	B/L ABSENT	NAD	NAD	NAD	NAD	10.9	27	NE	1.87	3.7	6.9	47	48	135	84	450	NEG	Y	B/L	Y	Y	N
Y	Y	N	N	AS	S	DECREASE	DULL	RT ABSENT	NAD	ENCEPH	NAD	NAD	9.8	42	NE	5.5	1.8	7.1	88	98	111	71	300	NEG	Y	RT	Y	Y	N
N	N	N	N	S	C	DECREASE	DULL	LT ABSENT	NAD	NAD	NAD	NAD	12.1	9	NE	0.95	2.9	5.5	23	12	56	99	112	NEG	Y	LT	N	N	N
N	N	N	N	AS	S	DECREASE	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.8	10	NE	1.21	3.6	7.1	141	121	87	56	200	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASE	DULL	RT ABSENT	NAD	NAD	NAD	NAD	9.4	8.8	NE	0.76	3.9	6.4	45	54	99	48	197	NEG	Y	RT	N	Y	N
N	N	N	N	AS	S	DECREASE	DULL	RT ABSENT	NAD	NAD	NAD	NAD	12	25	NE	0.56	4.1	7.8	33	32	101	35	113	NEG	Y	RT	Y	Y	N
N	N	N	N	AS	S	DECREASE	DULL	LT ABSENT	NAD	NAD	NAD	NAD	10	11	L	1.11	2.78	6.54	24	25	67	111	139	POS	Y	LT	N	Y	N

USG CHEST						CT CHEST	USG P.TAP		COMPLICATIONS			PLEURAL FLUID ANALYSIS													
LATERALITY	MILD	MOD	SIGNIFICANT	MULTIPLE LOCULATI	ON-SINGLE		DIAGNOSTIC TAP	THERAPEUTIC TAP	Y/N	HEMOTHORAX	H.PNEUMOTHORAX	HEMORRHAGIC	STRAW	PUS	OTHER	CHYLOUS	PROTEIN	GLUCOSE	LDH	PH	TLC	L	NE	L:NE	RBC
																	G/DL	MG/DL	U/L		CELLS/MM3	%	%		CELLS/MM3
LT	N	Y	N	Y	N	LT EMPYEMA	Y	N	N	N	N	N	N	Y	N	N	4.9	43	215	ND	2000	100	0		780
RT	N	N	Y	N	N	ND	Y	Y	N	N	N	Y	N	N	N	N	4.4	78	890	ND	150	80	20	4	500
B/L	N	Y	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	4.5	33	1500	ND	1000	20	80	0.25	10000
RT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	N	Y	N	N	5.1	5	1200	ND	990	20	80	0.25	2500
LT	Y	N	N	N	N	ND	Y	N	N	N	N	N	Y	N	N	N	2.8	89	200	ND	100	60	40	1.5	150
RT	N	Y	N	N	N	MOD RT P.E	Y	Y	N	N	N	Y	N	N	N	N	4.7	65	345	ND	200	20	80	0.25	430
RT	N	Y	N	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	3.6	78	230	ND	200	90	10	9	209
RT	N	Y	N	Y	N	ND	Y	N	N	N	N	N	Y	N	N	N	4.2	40	570	ND	600	25	75	0.3	450
LT	N	Y	N	N	N	ND	Y	Y	N	N	N	N	Y	N	N	N	5.3	96	200	ND	100	60	40	1.5	230

												DX	TB				M. P.E		EMPYEMA		PP.E				CHYLOTHORAX
PCV	AFB	GRAM STAIN	CBNAAT	C/S	M CELL	ADA	TG	OTHER	HISTOPATHOLOGY	TUMOR MARKER	AFB/PCR/MYCOBAC	EXUDATIVE P.E	P.E		TB EMPYEMA		PROBABLE	CONFIRME	SIMPLE	COMPLEX	TYPICAL	BORDERLINE	OMPLICATED	COMPLICATED	.
						U/L	MG/DL						PROBABLE	CONFIRMED	DEFINITIVE	PROBABLE									
ND	NEG	NEG	POS	NEG	NEG	129	N	N	N	ND	ND	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	POS	56	N	N	LUNG BX-ADENOCA	ND	ND	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	MRSA	NEG	54	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	P.AERU	NEG	45	N	N	N	ND	NEG	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	112	N	N	N	ND	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	39	N	N	N	ND	ND	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	NEG	NEG	78	N	N	N	N	ND	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
ND	NEG	NEG	NEG	MRSA	NEG	45	N	N	N	N	ND	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N
ND	NEG	NEG	NEG	NEG	NEG	99	N	N	N	N	NEG	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N

HEMOTHORAX	PULM.EMBOLISM	VIRAL	PARASITIC EMPYEMA	UNKNOWN	SPECIFIC TO ETIOLOGY		INTERVENT	TREATMENT							OUTCOME											
					ORGANISM	CARCINOMA		ABX	ATT	CHEMO	RADIO	SX	DELAY	OTHER	RESOLUTION OF SYMPTOMS			DAMA/EXPIRY			F/U CHEST XRAY			F/U CT CHEST		DX CHANGE
															AT D/C	1 MONTH	3 MONTH	AT D/C	1 MONTH	3 MONTH	AT D/C	1 MONTH	3 MONTH	1 MONTH	3 MONTH	1 MONTH
N	N	N	N	N	TB	N	PCD + STK	Y	Y	N	N	N	35 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	MILD	MILD	N	N	N
N	N	N	N	N	N	CA LUNG	N	Y	N	Y	N	N	27 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	MILD	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	Y	N	N	N	N	22 DAYS	N	Y	Y	Y	NA	NA	NA	PCD IN SITU	RES	RES	N	N	N
N	N	N	N	N	P.AERU	N	ICD + STK	Y	N	N	N	N	EXP	N	NA	NA	NA	EXP	EXP	EXP	NA	NA	NA	NA	NA	NA
N	N	N	N	N	TB	N	N	N	N	N	N	N	10 DAYS	N	N	Y	Y	NA	NA	NA	MILD	MILD	MILD	N	N	N
N	N	N	N	Y	N	N	N	Y	N	N	N	N	NO DEFINIT	N	Y	N	N	NA	NA	NA	MILD	RECURREN	RECURREN	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	15 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	MRSA	N	PCD + STK	Y	N	N	N	N	7 DAYS	N	Y	Y	Y	NA	NA	NA	MILD	RES	RES	N	N	N
N	N	N	N	N	TB	N	N	N	Y	N	N	N	35 DAYS	N	Y	Y	Y	NA	NA	NA	MOD	MILD	RES	N	N	N

E	COMPLICATION			TREATMENT CHANGE	
3 MONTH	AT D/C	1 MONTH	3 MONTH	1 MONTH	3 MONTH
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N
NA	NA	NA	NA	NA	NA
N	N	N	N	N	N
N	N	RECURRE	RECURREN	TH. P. TAP	VATS
N	N	N	N	N	N
N	N	N	N	N	N
N	N	N	N	N	N

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Date 20/4/2021

The Principal,
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Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Anu**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical profile of patients with exudative pleural effusion

Supervisor
Dr. Amit Bery
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The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, (if any).

S P Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

CLINICAL PROFILE OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

In

MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

NOVEMBER, 2019 DR. ANU

DEPARTMENT OF MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	Clinical profile of patients with exudative pleural effusion
For the degree of	M.D.Medicine
Name of the candidate	Dr. Anu
Supervisor	Dr. AmitBery Associate Professor, Department of Medicine, DMC&H, Ludhiana
Co-supervisor	Dr. Akashdeep Singh Professor, Department of Pulmonary Medicine, DMC&H, Ludhiana Dr. Dinesh Jain Professor, department of medicine DMC&H, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Collection of abnormal amount of fluid in potential space sandwiched between parietal and visceral pleural is pleural effusion.Increased hydrostatic pressure or decreased osmotic leads to transudative effusion, increased microvascular pressure due to disease of pleural or adjacent lung injury leads to exudative effusion. Based on modified lights criteria effusion can be classified as transudative or exudative. In this study we will focus on studying clinical profile of exudative pleural effusion and know its outcome.

Aim of Study: To study the clinical profile of exudative pleural effusion and know the outcome .

Materials & methods: A prospective study of 100 patients admitted in dmch will be carried out.

Clinical Significance: Exudative effusion has wide spectrum of etiologies which lead to pleural inflammation and pose diagnostic dilemma. Outcome of pleural effusion depends on underlying etiology, so it's important to make early correct diagnosis and start treatment so as to reduce morbidity and mortality.

Keywords: exudative effusion, clinical profile, thoracocentesis, outcome, lights criteria

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled"**CLINICAL PROFILE OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION**"exist in the Department of Medicine, Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected bythe candidate himself/herself under our supervision and guidance.

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APPROVAL PROFORMA
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Name of candidate	Dr. Anu
Department	Medicine
Topic of Thesis	CLINICAL PROFILE OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION
Likely date of appearing in PG examination	MAY, 2022
Date of enrollment	1st MAY,2019
Name of Head of Department	Dr. Dinesh Gupta
1) Supervisor **2) Co-Supervisor**	1) Dr Amit Bery 2) Dr Akashdeep Singh 3) Dr Dinesh Jain
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Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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4	DATE OF ADMISSION OF COURSE	1st MAY,2019
5	TITLE OF THE TOPIC	Clinical profile of patientswith exudative pleural effusion

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

List of causes of pleural effusion is quite exhaustive.Transudates are caused by small, defined group of etiologies[1], diagnosis of which is fairly easy as the underlying cause is clearly evident through history, clinical examination, and few common laboratory investigations.[2] Whereas exudative pleural effusion, produced by variety of inflammatory conditions requiring extensive evaluation.

A pleural effusion is collection of fluid abnormally present in the pleural space, usually resulting from excess fluid production and/or decreased lymphatic absorption.[3] Transudates- may be due to increased venouspressure (like in cardiac failure, constrictive pericarditis, fluid overload) or hypoproteinemia (cirrhosis, nephrotic syndrome, malabsorption).[4] Exudates are mostly due to increased leakiness of pleural capillaries secondary to infection, inflammation, or malignancy . Causes: pneumonia; TB; pulmonary infarction; rheumatoid arthritis; SLE; bronchogenic carcinoma; malignant metastases; mesothelioma; lymphangitiscarcinomatosis.[4]

Normal pleural fluid characteristics-clear ultra filtrate of plasma, ph of 7.60 to 7.64, protein less than 2% (1-2 g/dl), fewer than 1000 WBC per cubic millimeter,glucosesimilar to plasma, LDH of less than 50% of the plasma. Although a number of chemical tests have been proposed to differentiate pleural fluid transudates from exudates, the test first proposed by Light et al have become the criterion standards.[5] Exudative pleural effusions meet at least one of the following criteria, whereas transudative pleural effusions meet none (Light's criteria)-[1]Pleural fluid protein divided by serumprotein greater than 0.5. Pleural fluid LDH divided by serum LDH greater than 0.6. 3.Pleural fluid LDH greater than two thirds of the upper limit of normal serum LDH.[1] If one or more of the exudative criteria are met and the patient is clinically thought to have a condition producing a transudative effusion the difference between the protein levels in the serum and the pleural fluid should be measured, if this gradient is >31g/dl (3.1 g/dl), the exudative

categorisation by this criteria can be ignored because almost all such patients have a transudative pleural effusion.[6]

Symptoms (pain on inspiration and coughing) and signs of pleurisy (pleural rub) often precede the development of an effusion, especially in patients with underlying pneumonia, pulmonary infarction or connective tissue disease.[7] Other symptoms include cough, chest pain. The presence of chest pain, which results from pleural irritation, raises the likelihood of an exudative etiology, such as pleural infection, mesothelioma, or pulmonary infarction.[8] Other symptoms include edema, paroxysmal nocturnal dyspnea, night sweats, fever, weight loss, hemoptysis etc. Past history of trauma, tuberculosis, malignancy, diabetes mellitus, rheumatological disease etc, treatment history, occupation as well as family history of any medical disease is important. On examination- Dullness to percussion, decreased tactile fremitus, and asymmetrical chest expansion, with diminished or delayed expansion on the side of effusion: These are the most reliable physical findings of pleural effusion.[9,10] Position of trachea is noted. Auscultation over the pleural fluid characteristically reveals decreased or absent breath sounds.[1] Other findings: lymphadenopathy, ascitis, edema. Investigations- basic blood investigations, Sputum analysis, Chest X-ray, USG chest are done, CT chest if indicated. Diagnostic thoracocentesis is done. Ultrasonography guidance at bedside significantly increases the likelihood of obtaining pleural fluid and reduces the risk of pneumothorax.[11,12] Pleural fluid biochemical & cytological analysis, culture, ada, LPA/CBNAAT, cholesterol and triglyceride levels, amylase, fluid for malignant cytology, immunological studies are done. Usg/ct guided pleural biopsy, VATS, bronchoscopy are done wherever indicated. Treatment depends on the underlying etiology. Therapeutic thoracocentesis allows re expansion of lungs. Surgical treatment, sclerosing agents, pleurodesis as in recurrent effusions due to malignancy, TPC (tunneled pleural catheters) can be used.

6.2 REVIEW OF LITERATURE

A prospective study conducted by PP Prabhudesai and et al in Maharashtra (1993) on exudative pleural effusions in patients over forty years of age –analysis of seventy six patients indicated that malignant pleural effusions were the most common in this series, found in 49 patients (64.47%), all but one being metastatic from elsewhere. Forty were secondary to a carcinoma of a bronchus, 3 from carcinoma of breast, 1 each from the carcinoma of ovary, esophagus, and larynx; lymphoma accounted for the remaining 2. Infective causes accounted for 24 of the effusions (31.57%). Of the infections, tuberculosis was the most common, accounting for 17 of the 24. Other infective causes included bacterial empyema in 4, ruptured amoebic liver abscess in 2, and actinomycosis in 1. Pancreatitis, pulmonary thromboembolism, and a post- craniotomy syndrome were diagnosed in 1 patient each, while the diagnosis remain unknown in the remaining 5 patients. In 2 patients the diagnose was made on autopsy.[13]

A study by Luis Valdes and et al (2010) Spain on Diagnosing Tuberculous pleural effusion using clinical data and pleural fluid analysis: a study of patients less than 40 years old in an area with high incidence of tuberculosis was conducted. Results were model 1 selected two variables (ADA > 35 U/L) and lymphocytes (>31.5%) and correctly classified 216/218 effusions (1 false negative, 1 false positive). Model 2 (without ADA) selected 3 variables: lymphocytes (>31.5%), fever and cough and correctly classified 207/218 effusion (8 false negatives, 3 false negatives). The sensitivity of models 1and 2 was 99.4% and 95.2%, specificity 98.1% and 94.3% and accuracy 99% and 95%.[14]

A study conducted by Rohit Rungta and et al in 2013 on Comparative analysis of pleural fluid biochemical parameters with cholesterol to differentiate transudates from exudates suggested that, according to their etiology, 7 of the effusions were transudates and 49 were exudates. Using a cutoff point of 45 mg for pleural cholesterol and values for protein and lactate dehydrogenase (LDH) of Light *et al.*, the best diagnostic power corresponded to the combination of pleural

cholesterol and LDH; cholesterol level over 45 mg/dL and/or LDH over 200 IU/L differentiate exudates from transudates with a sensitivity of 99% and a specificity of 98%.[15]

Another study- Study of 100 cases of pleural effusion with reference to diagnostic approach was conducted by Pujan Parikh and et al in Gujarat (2016) indicated that most of the patients were between the age group of 31-40 years and males (68%). Most of the patients of tuberculous effusion were from younger age group between 21-50 years. Most common symptom was chest pain (72%) followed by fever (62%). Most of cases were tuberculous (62%) followed by malignant (18%). There were 15 patients with undiagnosed pleural effusion, in which, thoracoscopic pleural biopsy was done, among them 9 patients had malignancy and 5 patients had tuberculous pleural effusion.[16]

Another study was conducted by Biswajit Biswas and et al (2016) Pleural effusion: Role of pleural fluid cytology, adenosine deaminase level, and pleural biopsy in diagnosis. It was found that Tuberculosis was the commonest non-neoplastic lesion followed by chronic nonspecific pleuritis comprising 60% and 33.3% of the non-neoplastic cases respectively and tuberculosis was predominantly diagnosed in the younger age group. Majority (70.8%) of malignancy cases were in the age group of >50-70. Adenocarcinoma was found to be the commonest (66.7%) malignant neoplasm in the pleurae followed by small-cell carcinoma (20.8%).[17]

A Study conducted by Saha K and et al in 2017 in West Bengal on demographic , clinical , biochemical and etiological characteristics of malignant pleural effusions from Eastern India identified that out of 166 patients, 72.89 % were males and 7.11% were females. Mean presentation among males was 64.3 ±12.7 and among females was 52.5 ±14.8. Most common presenting symptom was dry cough (87.9%) and most common presenting sign was clubbing (54.5%). Massive effusion was found in 45.785 of cases. Pleural fluid macroscopic appearance was hemorrhagic in 54.82% cases. Mean adenosine deaminase activity in MPE was 24.05 U/L. Mean pleural fluid/serum protein ratio was 0.65, mean pleural fluid/serum lactate dehydrogenase

ratio was 1.01. Most of the cases (84.94%) were diagnosed by pleural fluid cytology for malignant cells. Primary cancer was diagnosed in 136 (81.93%) cases; among which 121(88.97%) causes were lung cancers, among which adenocarcinoma (52.89%) was most common histology.[18]

A study was conducted by Rahul Gupta and et al (2018) in Jammu and Kashmir named Spectrum of pleural effusion etiology revisited in 18–70 years of age group: A tertiary care center-based study of 1000 patients. It was found that out of total 1000 patients, 69.5% had tuberculosis followed by malignancy (16%) with the systemic causes forming about 15% bulk of the patients with pleural effusion. It was found more in males, associated with smoking, and majority of patients had unilateral effusion. Eighty-nine percent of patients had exudative effusion.[19]

A study conducted in West Bengal (2018) by Maikap MK, et al on Etiology and clinical profile of pleural effusion identified that the most common cause of pleural effusion in this study was tuberculosis (68.8%), followed by malignancy (14%), empyema (6%), and transudative effusion (2.8%) pleural effusion was commonly seen in males (66%).the occurrence of pleural effusion was maximum in the age group 21-30 years, but malignant pleural effusion was more common above 60 years of age. Right sided effusions were more common. Estimation of pleural fluid deaminase plays a significant role in the diagnosis tubercular pleural effusion. Pleural fluid cytology and closed pleural biopsy can diagnose most of the cases of pleurai effusion due to malignancy.[2]

S Laxma Reddy and et al conducted a study published in 2019 named Clinical and etiological profile of an exudative pleural effusion in a tertiary care center at Hyderabad, Telangana .Among the study participants, 40 were male and 23 were female patients with male-to-female ratio of 1.7:1. Mean age of the study population was 48.8 ± 18.7 years. It was found that the most common presenting symptom was dyspnea (84%) followed by cough (80%), fever (65%), and chest pain (43%). The most frequent cause of pleural effusion was tuberculosis in 38% of patients, followed by parapneumonic effusion (28.5%) and

malignant pleural effusion (22.2%). Three patients had chylothorax, two patients had pancreatic pleural effusion and the diagnosis was unknown in two patients. Mean ± standard deviation (SD) adenosine deaminase (ADA) value of the study population was 45.3 ± 28.1. Mean ± SD of ADA values in tuberculous, parapneumonic, and malignant pleural effusion was 54.5 ± 16.8, 65.2 ± 30.7, and 18.2 ± 11.0, respectively.[20]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical profile of patients with exudative pleural effusion.
- To study the outcome of patients with exudative pleural effusion.

7. MATERIALS AND METHODS

Study will be carried in 100 admitted patients in Dayanand Medical College and Hospital, Ludhiana.

7.1 Source of Data

100 patients with exudative pleural effusion admitted in DMC&H will be enrolled in this study. Diagnostic/therapeutic guided thoracocentesis will be done. Patients who are diagnosed with exudative pleural effusion will be included in this study. They will be followed at 1 month and 3 month of discharge. Their demographic, etiological, clinical, radiological profile and outcome data will be recorded and analysed.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA

- age>18 years,
- Aspirable pleural effusion
- Meets the lights criteria for exudative fluid

EXCLUSION CRITERIA

- age<18years,
- non aspirable pleural effusion,
- transudative pleural effusion
- Consent not given by the patient

NUMBER OF PATIENTS- Study will include 100 patients with exudative pleural effusion.

OUTCOME- To study the clinical response and chest radiography at 1 month and 3 month of treatment initiation, or any re- revision of diagnosis at 1 month and 3 month.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively.For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test was used when the

expected frequency is less than 5. Receiver operator characteristics (ROC) curve was done, and criterion value will be estimated depending on the specificity and sensitivity. Area under curve (AUC) will be measured. A probability value (*p* value) less than 0.05 will be considered statistically significant. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes, the study requires basic investigations like CBC,LFT, RFT, chest X ray , ultrasound, CECT/HRCT chest, ESR, serum ldh ,pleural fluid analysis which will include– fluid glucose, protein, ldh, , amylase, hematocrit fluid, triglyceride, cholesterol, cytological examination, ada levels, pcr- line probe assay/CB NAAT, gram stain, culture , and, fluid for malignant cytology. Immunological test- RA factor, TSH, ANA IF/profile, complement levels, tumors markers wherever indicated.

Following interventions will be required - usg guided pleural tap, fine needle aspiration cytology and biopsy (usg/ct guided) wherever indicated, bronchoscopy/thoracoscopy wherever indicated.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOURINSTITUTION IN CASE OF 7.3?

Attached here with

8. LIST OF REFERENCES

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PROFORMA

Sr. No____

NAME-	AGE/SEX-
DATE OF ADMISSION-	DATE OF DISCHARGE-
MRD NO-	OCCUPATION-
ADDRESS-	SOCIO ECONOMIC STATUS-

HISTORY	SYMPTOM	Y/N	DUR	SYMPTOM	Y/N	DUR
PRESENT HISTORY	CHEST PAIN			FEVER		
	SOB			CHEST TRAUMA		
	COUGH			HEMOPTYSIS		
	EXPECTORATION			OTHER		
PAST HISTORY	TB			CLD/ALD		
	HTN			CKD		
	DM			OTHER		
EXPOSURE TO DRUGS/TREATMENT	NIROFURANTOIN			MTX		
	AMIODARONE			OHT		
	PHENYTOIN			RADIOTHERAPY		
	OTHER					
PERSONAL HISTORY	SMOKING			MENSTRUAL HIS		
	ALCOHOL			STD EXPOSURE		
	OTHER					

GENERAL PHYSICAL EXAMINATION ON ADMISSION

APPEARANCE-	SENSORIUM-	HT-(m)	WT-(kg)-	BMI-(kg/m^2)
TEMPERATURE-	PAIN-	HR	RR	BP-
PALLOR-	ICTERUS-	CYANOSIS-	LAP-	
CLUBBING-	JVP-	EDEMA-		

SYSTEMIC EXAMINATION ON ADMISSION-

CHEST	SYMMETRY-	TRACHEA-	APICAL IMPULSE-
	TACTILE FREMITUS-	PERCUSSION-	PLEURAL RUB-

Signature of Candidate **Signature of Supervisor**

	AUSCULATION-		
CVS			
CNS			
PER ABDOMEN-			
ANY LOCAL FINDING			

INVESTIGATIONS

HB	PCV-	TLC-	DLC-	PLATELETS
CREATININE-	NA/K -	S. ALBUMIN-	S.PROTEIN	PTI INR
UREA-	RBS-	BILLIRUBIN(T/D)-	ALT/AST-	ALP-
ESR	MONTOUX TEST	URINE:PROTEIN GLUCOSE DEPOSITS-	S. LDH-	TSH/T3/T4-

RADIOLOGICAL INVESTIGATIONS/FINDINGS & INTERVENTIONS

CHEST X RAY				
USG CHEST	LATERALITY-			
	QUANTITY-	MILD	MODERATE	SIGNIFICANT
	SEPTATIONS-			
	LOCULATION-			
CECT/HRCT CHEST				
US GUIDED THORACOCENTESIS-	DIAGNOSTIC / THERAPEUTIC AMOUNT OF FLUID ASPIRATED -			

Signature of Candidate **Signature of Supervisor**

INTERVENTIONS

US GUIDED THORACOCENTESIS-	DIAGNOSTIC/THERAPEUTIC-
	AMOUNT OF FLUID APIRATED-
POST PROCEDURE CHEST XRAY/COMPLICATIONS -	

PLEURAL FLUID ANALYSIS-

MACROSCOPIC-	COLOR- HEMORRHAGIC/STRAW/PUS
	OTHER-

SAMPLE	PROTEIN	GLUCOSE	LDH	PH	OTHER
P.FLUID					
SERUM					

TLC-	DLC-	RBC-	PCV-	AFB-
PCR-	C/S-	M CELL-	ADA-	MISC-

ANALYSIS-

OTHER INVESTIGATIONS (if indicated)

ANA(IF)-	RA FACTOR-	ANTI CCP
ANA (PROFILE)-	C3/C4 LEVELS-	MISC

OTHER INTERVENTIONS /PROCEDURE–

Signature of Candidate

Signature of Supervisor

FINAL DIAGNOSIS-

OUTCOME

TREATMENT	DATE OF INITIATION	DURATION OF SYMPTOMS	DELAY IN INITIATION FROM 1ST SYMPTOM-
ANTIBIOTICS			
ATT- CATEGORY			
CHEMOTHERAPY/RADIOTHERAPY			
SURGICAL MANAGEMENT			
OTHER			

FOLLOW UP

	AT DISCHARGE	1 MONTH	3 MONTH
RESOLUTION OF SYMPTOMS			
CXR PA VIEW			
ADVERSE OUTCOME/ COMPLICATION/ OTHER			

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"CLINICAL PROFILE OF PATIENT WITH EXUDATIVE PLEURAL EFFUSION"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"CLINICAL PROFILE OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION " ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o..... .....am willing to participate in the study **"CLINICAL PROFILE OF PATIENTS WITH EXUDATIVE PLEURAL EFFUSION"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**